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PART I.

Original Contributions.

CLINICAL GROUPS OF CASES OF AMAUROSIS.

By JONATHAN HUTCHINSON.

IN investigating the extremely complicated questions involved in the subject of amaurosis (using the term in a very wide sense, as embracing cases of atrophy of the optic nerves from all causes, as well as cases of blindness without visible change), it is most desirable to have a large material to work upon. Without in the least disparaging the importance of the minute study of individual cases, both in regard to life-history and pathological anatomy, we may, I think, make some use of our less complete records by throwing them together in large numbers, and attempting to subdivide them into classes in accordance with their leading clinical features.

Even a very moderate diligence in case-taking supplies, in the course of years, an accumulation of material which

rapidly becomes most unwieldy. The memory is taxed to the utmost to retain even the chief points of the more remarkable cases, and all details gradually slip from its grasp. The recurrence to note-books and re-perusal of manuscript is most laborious, and to many almost impossible of satisfactory accomplishment. Each succeeding day brings as much new matter as it is practicable to deal with, and thus that which has been collected in the past becomes but too often buried and forgotten. Yet it is only by the careful collation and comparison of cases that the art of clinical observation can advance. It has often occurred to me that in facing this difficulty, and source of loss to science, something might be done if observers would record their cases not in full, but in brief abstract. It is often possible, in a few lines, to sum up the chief facts which are recorded in many pages of manuscript, and give them, also, in a manner which shall, to those familiar with the subject, suffice for most purposes. Such records in abstract will often enable the author to classify his facts with tolerable correctness, and give the reader a good opportunity of estimating their character and significance.

By this plan, we also gain much security against the risk of overlooking important facts. With the whole material before us, we proceed, as a botanist would do with his dried specimens, to put into groups those of which the names are known, and then investigate more closely those which remain over. When this is done, it will frequently be evident that of the cases which remained peculiar or, perhaps, isolated, two or three closely correspond with each other, and thus, by degrees, groups are formed and natural affinities brought out. I make these remarks somewhat in the way of apology for bringing before the reader such a large collection of material, much of which may appear somewhat crude.

In the present paper short outlines only are given of a considerable number of cases of which I have long notes. Many of them are incomplete in one or more respects, but

none are included which did not seem full enough to be useful for purposes of comparison or confirmation.*

Group I. Optic Neuritis from Tumour in the Brain.

In this group I have but six cases to mention, two of which I have already published in detail. In four of these the diagnosis of "tumour" as distinct from syphilis on the one hand, and tubercular meningitis on the other, was almost certain. In three, indeed, an autopsy was made. In Case 2, the diagnosis was less certain, and it is possible that it may have been an instance of recovery from tubercular meningitis and subsequent relapse.

Intense headache; neuritis, with "choked discs," and subsequently atrophy; absolute blindness, and dilated pupils; convulsions; frequently paralysis of other orbital nerves, or some degree of hemiplegia; such are the principal symptoms which occur in amaurosis from cerebral tumour. From tubercular meningitis, we may diagnose by the circumstance that the symptoms develop more gradually, are attended by far less constitutional disturbance, and are more protracted. The fact that no benefit accrues from treatment may help us to distinguish them from cases of syphilitic gummata. It is never safe when this suspicion is entertained to rely upon the absence of history of syphilis. Many warnings on this point have occurred to me, and so similar in many cases are the symptoms of tumour and those of gumma, that I would incline, in almost all cases, to let the patient have the chance of benefit which the trial of iodide of potassium in full doses affords. The neuritis which attends gumma rarely sets in with the same sudden-

* Those of the following cases which have been published before, may be identified by the references:—

M. C. T. = Medico Chirurgical Transactions.

L. H. R. = London Hospital Reports.

O. H. R. = Ophthalmic Hospital Reports.

The letter B refers to cases under care many years ago, for the most part prior to 1864.

ness and severity as that caused by tumour, and rarely results in such absolute blindness.

I have, of course, seen many other cases in which, with optic neuritis, tumour was suspected; but in most instances they have been transferred to my medical colleagues, or have remained under care only a short time.

Case 1. F. 202. Eliza D., 14. (Full details).

Double optic neuritis, with but slight defect of V.; and paresis of R. 6th nerve.

Previously severe headache. Subsequently severe headache and some fits, and stupor. Partial hemiplegia.

Death rather sudden about 3 years after commencement of symptoms, æt. 17. *P.m.*, two tumours, one at base of skull as large as a hazel nut and almost cartilaginous, much serous effusion, the other in optic thalamus.

Her father died of general paralysis of the insane æt. 38. At the p.m. "the usual appearances of that disease in the brain," the other organs healthy. A cousin idiotic, and other relations suffering from nervous disease.

Case 2. F. 281. Maria B., 11. (Full details).

Atrophy after neuritis, with absolute blindness, after an illness with cerebral symptoms; weakness, severe frontal headache, loss of appetite and sickness. Ps. partly dilated.

Later; nystagmus, another illness with fits, ending fatally at about æt. 12—13. *No p.m.*

Case 3. B. p. 42. Frances Montgomery, 24, single, came to me in June, 1862, totally blind with profuse neuritis. Her sight had begun to fail about 2 months previously, and had progressed to total blindness in about 6 weeks.

She remained totally blind, with wide pupils, which contracted a little when exposed to light.

She became feeble and nervous rather before the time when sight began to fail, and had to leave her place on account of weakness. She also became subject to severe headache at back of head, keeping her awake.

She was under care at the Metrop. Free Hosp. for 3—4 months and continued to get feebler and thinner, and still suffered from

much headache and some sickness. She had, however, no paralysis and no loss of sensation. She died just a year from the beginning of her symptoms. *No post mortem. Diagnosis, cerebellar tumour.*

Case 4. G. 171. Emma T., 9.

Paralysis of one 6th N., followed by optic neuritis of same, and later of other eye.

Probably tumour.

Case 5. O. H. R., v, 163. Mrs. W., æt. 36. (Full details.)

Neuritis as an accompaniment of other cerebral-nerve symptoms in a case of tumour of crus cerebri. Autopsy.

Case 6. O. H. R., v, 167. B. 267 and 382. Mary Anne A., 20.

Neuritis and almost complete amaurosis. Cerebral tumour diagnosed. No paralysis; very severe headaches; torpor; sluggish pupils; partial insanity.

(No notes of *p.m.*)

Case 7. Stephen W.

For a case of well marked neuritis from cerebral tumour, see that of Stephen Watson (Lond. Hosp., 1871, p. 306. Published in Brit. Med. Jour., Feb. 19, 1872).

Group II.—Amaurosis from Optic Neuritis in the Tertiary Stage of Syphilis.

The evidence of syphilis was not conclusive in all the cases which I have included in this group, but in all it was sufficient to warrant a tolerably confident suspicion. The group is an exceedingly important one, since the cases are frequent, and much may be done by treatment for the patient's benefit. Of the cases which ended in blindness, it was not known in any that efficient treatment had been resorted to in the neuritis period. The group comprises 21 cases. Two only were known to have died, and of these in one the diagnosis had been "cerebral tumour" and the treatment inefficient. I brought this important case (No. 16) before the notice of the Pathological Society,

and it is recorded in this year's volume of Transactions. In only one case (23), was the disease believed to have been inherited, a fact of much importance in reference to the rarity of all forms of cerebral syphilis in the subjects of hereditary taint. Even this single case was not an undoubted instance of cerebral gumma, although that diagnosis may be admitted to be very probable.

As regards sex, it is of interest to note that exactly half were women, or, including the hereditary case, 11 women to 10 men. As it is probable that many more men than women suffer from syphilis, this proportion is somewhat unexpected.

The cases included under the term "Cerebral Syphilis," must be divided into three or four different classes. 1st, those in which disease of the coats of arteries lead to thrombosis, or hæmorrhage; 2nd, those in which gummata of the meninges are present; 3rd, those in which disease of the bone exists; and 4th, those in which the changes implicate nerve-centres, but are attended rather by degeneration than growth; and lastly, perhaps, a group in which a travelling form of primary neuritis is present. I speak, of course, somewhat conjecturally in admitting the two last, but clinical facts support the probability. Of these five groups, it is probable that the 2nd supplies us with by far the larger proportion of amaurosis cases. The neuritis which occurs in the tertiary stage of syphilis, is usually secondary to gummata in the meninges. Arterial disease, with hæmorrhage or softening, may occasionally cause neuritis, but whilst arterial disease is, perhaps, the commonest form of "cerebral syphilis," we meet with it but rarely as a cause of defect of sight. I have not admitted into the present group any cases which obviously belong to the 4th or 5th classes, since I wish to reserve them for special consideration subsequently. It is possible, however, that Case 26 ought to have been reserved on this score, for the only symptoms of gumma or of bone disease were headaches, and there was no proof

of neuritis. In nearly all the others there was reason to suspect gummata, and in very few was there the history of the sudden attacks of paralysis which denote arterial disease. A glance through the cases will show that the neuritis which occurs with cerebral gumma varies a good deal in different cases as regards its symptoms and rate of development. In some (as in Cases 17 and 18), both eyes failed together, and rather rapidly; but in many others, the progress was more like that of Case 20, in which first one and then the other was affected, and progress, both as regards advance and cure, was gradual. Severe headache stands out in almost every case as the most conspicuous symptom, and with it we have usually giddiness and occasionally deafness, convulsions, paralysis of other nerves, &c. In one instance in which the history of symptoms in the patient was wholly wanting, the woman had borne a syphilitic child.

Cases 17 and 18 are examples of the rapidly-triumphant results of treatment, both patients having regained perfect vision in a few weeks under the use of the iodide. In Case 20, the result, although much more gradual in attainment, was scarcely less satisfactory. A large majority of the patients came under my care when the atrophy was established and treatment hopeless.

Case 8. G. 17. Thomas W., 50. Syphilis probable, but not proved.

Atrophy of discs and amaurosis, following neuritis.

History of numerous convulsive seizures without loss of consciousness, several years ago.

Subsequently sudden L. hemiplegia, and death in about a month. No *p.m.*

Case 9. G. 56. Mrs. P., 39.

Double recent optic neuritis and white dots with hæmorrhages, with absolute blindness. Blindness coming on suddenly during or directly after a fit, 3 weeks ago.

History of sickness and headache for several months before the fit.

Note 4 years later; blindness permanent; has had another fit.

No history of syphilis, but of a node on forehead following a blow had ulcerated and left a scar.

Case 10. G. 268. Isaac C., 44.

Failure of V. after 2 months of headache and sickness, for which he had to keep in bed. Failure rather rapid, and then remaining stationary at no p. l. till admitted about a year later.

Atrophy of discs and diminution of arteries, with remains of neuritis.

Some periosteal swellings on skull.

Probably neuritis from intracranial nodes or gummata.

Case 11. G. 87. Eliza D., 25, unmarried.

Atrophy of discs after neuritis, with much diminished vessels; absolute blindness of L. and shadows with R. No choroiditis. Ps. fairly active, but L. larger than R.

Failure of V. 5 months (Christmas, 1871), preceded for 6 months by severe pain in head and vomiting (June, 1871). That illness preceded immediately by "rheumatic fever" of 3 months' duration, with swelling and great pain in joints and limbs (? nodes) (spring of 1871). Scar of cellular node in neck.

History of severe secondary syphilis, with double iritis (synechiæ) at æt. 15, *i.e.*, 10 years before the cerebral symptoms and neuritis.

Case 12. F. 365. Charles H., 35.

Double optic neuritis with hæmorrhages. V., R. 16 J. L. 2 J. and $\frac{20}{20}$.

Failure of V. about 4 months, preceded by intense headaches; paroxysms lasting a few hours and coming on every 2 or 3 days, relieved by lying down. They ceased after leeching the head, and had ceased when V. began to fail.

History of several attacks of giddiness and of a fit, several months before the headache began.

Probably had syphilis several years ago, but not proved.

Case 13. O. H. R., v, 109, B. 344. Mrs. A., 30.

Double optic neuritis, going on to all but complete blindness in about 12 months, preceded by sickness, and accompanied by severe headache.

Discs gray-white, quite flat, without much opacity. Vessels of fair size.

Syphilis fully from husband, 4 years before failure of V.

"For years" subject to pain in occiput, giddiness and noises in ears.

At one time had weakness of L. limbs.

Loss of smell occurred with the amaurosis. Hearing perfect.

Case 14. B. 192. Mrs. H. S., 31 (1864).

Neuritis (no great amount of opaque effusion) and extreme defect of sight in a woman, the subject of tertiary syphilis, and who had suffered severely for the last 8 years from nodes on the skull and legs, and from various symptoms depending on affection of nerves of special sense. Taste, smell, sight, and hearing, all more or less affected about the same time. Failure of sight varied on different days. Accompanied by intense headache.

No note of progress.

Case 15. B. 46. Charles W., 29.

1855 or 1856. Had chancre and rash.

1858. A six weeks' illness, with much pain in forehead and eyes, and repeated vomiting. Then had diplopia, soon followed by failure of V., first in one eye, then the other. V. became worse during a salivating course of mercury. Also took iodide largely, without getting better or worse.

1860. Under me for first time. R. eye diverged rather. Ps. active; R. rather larger than L. V. about 20 J.

1862, æt. 29. Atrophy of *o.ds.*, with partial absorption of choroid near them; in the R. this passes nearly to *y.s.* Edges of discs jagged; arteries small. V. about same; no worse. Ps. dilated, but freely movable.

1865. V. same. R. p. l.; L. barely 18 J. Ps. dilated and sluggish, but act a little. Health good. No other syphilitic symptoms.

Case 16. B. 272 (and O. H. R., v, 170). Eliza Jane T., 36.

Neuritis, with history of variations of sight, ending after a few months in total blindness of one eye and all but similar state of the other (occasionally sees shadows).

Preceded for a few months by severe pain in head and epileptic

fits (no tongue-biting), which have since become frequent; history of partial loss of intellect since the fits began.

Probable syphilis 4 years ago (scar-leaving rash).

Case 17. Mrs. B. See Pathol. Transactions, 1877.

Double optic neuritis, with intense headaches and partial amaurosis. Recovery of fair V.

Four years later, relapse of the very intense headaches, but no failure of V. Temporary aphasia, and afterwards weakness of R. side.

Death. Gummata of dura mater, and thickening of this membrane, and condensation of bones of skull.

Syphilis certain, but the date unknown.

Case 18. Mr. M., about 30.

Double optic neuritis with almost complete amaurosis and intense headaches. Rapid recovery of perfect V. under use of iodide.

Syphilis, with very prolonged secondary and intermediate symptoms, about 12 years before the neuritis.

Since his recovery from the neuritis now five years ago, Mr. M. has often been under my care for headaches, giddiness, &c., and is always cured by the iodide. He has had no relapse of neuritis, and now sees perfectly.

Case 19. Mr. T. had syphilis probably about 15 years before I saw him, on May 1st, 1874.

He had then been failing in health for some months, the chief complaint being a sense of faintness, and pain in the back of the head and neck, "a dull steady pain." There had also been shooting pains in the limbs. When I saw him his sight was very defective, and there was neuritis.

After a short course of iodide, the discs cleared all but perfectly, and he regained perfect vision.

Case 20. Edward J., 31.

Double optic neuritis, with gradual failure of V. for several months. Recovery of nearly perfect V. while taking iodide (but taking it irregularly).

Node in thigh and syphilitic ulcers of legs during the neuritis.

No note of head symptoms.

Case 21. Mr. R. G. W., æt. 36.

Optic neuritis of L., followed by same condition in R. after a considerable (?) interval.

Partial atrophy of L., resulting in a condition very like that from tobacco, but with great defect of V.

He had had syphilis, with prolonged secondary stage, 15 years before the neuritis. He suffered at the date of the neuritis from giddiness, ringing in ears and headache. He recovered under treatment.

Case 22. Edward Q., 35.

Atrophy of discs with amaurosis of 4 years' duration. Failure rapid, in one week.

Opaque white discs, with slight haze of edges, and some notching. Arteries diminished.

Possible syphilis 12 years ago.

Case 23. Lond. Hosp., 1871, p. 23. Sarah B., 44.

In the Hospital for superficial lupus of face (8 years' duration), with scarcely any ulceration ("Bat's wing"), February 17, 1871.

She also had white atrophy of discs with some (but not extreme) diminution of retinal arteries; the margins of the discs were irregular, as if from former neuritis. (There is unfortunately no note of her vision, but from other statements it is probable that she could see fairly.)

Fifteen years ago she lost dead bone from her nose and from the roof of her mouth. This was followed about 2 years later by what she calls "St. Vitus's dance," and soon after by double vision and dimness of sight. The sight got better, but she next had partial paralysis of the left side; a permanent and indeed increasing weakness of left leg has resulted, and she now cannot walk without a stick. She also gives the history of an illness attended by 6 *weeks*' loss of consciousness; this was a few months after the paralysis of left side. Her entire train of nervous symptoms would appear to have come on within about 1 year or $1\frac{1}{2}$ year. She was under the care of Mr. Bowman and Dr. Todd, and was afterwards at St. Thomas's Hospital.

Married 19 years (4 years before the necrosis); 2 pregnancies, both ending in still births.

Case 24. *Neuritis ending in total Blindness in one Eye, and all but complete Blindness in the other, during Pregnancy, in a Young Woman, suspected to be the subject of Inherited Syphilis.*

Lond. Hosp. 1871, p. 254. Hannah T., 22, married.

For 12 months past had been subject to fits, with loss of consciousness. The first occurred when she was 6 months pregnant, and sight began to fail soon after it. *She was unconscious for* 3 *days.* Very numerous fits since then, sometimes 3 in a week. The fits often preceded by excessively severe frontal headache, generally beginning in the morning, and accompanied by vomiting. The headaches sometimes prevent her from recognising people. She often cries out with the pain, and cannot keep her head still. Her fingers and feet "seem to be all on the work" before she has a fit. The headache generally culminates in a fit, and is relieved afterwards.

She has had many cellular and some periosteal nodes. Her aspect and teeth are not quite typical. Iodide relieved the pain and cured the fits during the 10 months that she was under care, but sight remained just the same. The discs cleared, and became almost clean, but they were much atrophied, and she remained nearly blind.

It is possible that the symptoms may have been related to pregnancy. Her urine contained no albumen. (Full details).

Case 25. *Neuritis accompanied by Paralysis of both* 6*th Nerves, one* 4*th Nerve* (*?*), *superior and internal branches of one* 3*rd, and the iridal fibres only of the other* 3*rd Nerve—Tertiary Syphilis probable.*

Lond. Hosp. 1871, p. 339. Elizabeth F., 42, a married woman, began to suffer from pain in the head (chiefly nocturnal), accompanied almost from its commencement by dimness of sight and by some muscular defect of the L. eye, causing it to "turn up."

She came to Moorfields 3 months after these symptoms began, and was admitted as an in-patient, but there are no notes of progress. Her optic discs were then pale, and showed remains of neuritic exudation.

It is probable that she and her husband had syphilis 12 years previously. 3 children had been born prior to the date of her (admitted) venereal disease; all are living and well. Of

6 born since, only the youngest is living, 3 of the number having died within 3 months of birth, and 2 within 2 years old; all these 6 had "thrush."

Case 26. G. 225. Mrs. T., 35.

Failure of R. about 8 years.

„ L. „ 3—4 years.

Advanced atrophy of R., but with still some swelling and remains of lymph.

Neuritis passing to atrophy in L.

Ps.—L. very small; R. larger and motionless to light.

She has borne many dead fœtuses, and her only living child is very markedly syphilitic, and also has defective sight in one eye from opacity in the vitreous, and some pallor of discs. No direct evidence of syphilis in the patient.

Case 27. C. 70. Mrs. C., 27. Symmetrical failure of sight, without ocular changes. ? Descending neuritis. Syphilis rather severely seven years ago. No reminders until six months ago, when severe headaches began. No other cerebral symptoms. Discs at first normal, but artery small. Under observation 1 month, and the discs got a very little paler. Snellen 70 at 1 foot.

Case 28. F. 49. Thomas K., 55.

Gradual failure during about 1½ year, down to 19 or 20 J. with R. and p. l. with L. He had some giddiness, and for a time "dreadful" headaches, sometimes making him delirious; was out of his mind ("stupid") for a short time, 2—3 years after failure of V. V. began to fail about æt. 47.

Æt. 51. Discs atrophied, and some doubtful haze from former neuritis. Arteries not diminished materially. Ps. normal size, but motionless.

Æt. 55. Discs same; more atrophied on *y.s.* sides. White lines along vessels. Neuritis doubtful.

Health good. No paralysis. No headache now. Occasional numbness of L. upper extremity.

Syphilis denied, but probably the cause. History of sudden deaths on father's side.

Group III.—Neuritis with Cerebral Illness in Children ending in perfect Recovery of Health.

Tubercular meningitis is a common cause of optic neuritis, but the cases usually end fatally, and do not proceed to that stage to which the term amaurosis is applicable. In a certain number, however, after a severe attack of cerebral symptoms, the child recovers, and on recovery is found to be blind. Of these a considerable proportion find their way to ophthalmic surgeons. It is of such cases that the following group is composed. It is possible that in some, the diagnosis of tubercular meningitis could not be sustained, and that they ought, rather, to be regarded as examples of arachnitis at the base of the brain, implicating chiefly the sub-arachnoïd spaces. The precise nature of the primary lesion must be left in doubt. The fact, however, remains, that a large number of such patients recover and regain excellent health, remaining usually quite blind. Now and then, after long periods of blindness, slow improvement sets in, whilst in a small minority fatal relapses of cerebral symptoms occur.

I wrote a paper on this subject, and recorded a series of cases in this Journal (vol. v, p. 310), some years ago. Most of these cases are briefly referred to again in the following list. It is possible that in some of the cases the disease may have been of the nature of cerebro-spinal meningitis, and in others it is probable that the head-symptoms were in association with one or other of the exanthemata.

Case 29. C. 208, William W., 7.

Gradual failure of V. for 6 months, without headache or sickness.

Six months after it began, neuritis of each disc passing into atrophy, with choroidal atrophy at *y.s.*, "as in the other cases."

Ps. of usual size and fairly active.

Case 30. O. H. R., v, 311, C. 125. Mary C., $6\frac{1}{2}$.

Amaurosis with atrophy of discs; some haze, and much diminution of arteries and veins. Changes more advanced in L. than R.

In L. also, minute white dots in choroid or retina, chiefly in a group near *y.s.* Some slightly pigmented.

L. believed to have been as bad as now since æt. 12 months.

R. failing only 2 weeks, according to statement.

Case incomplete.—? Heredito-syph. choroido-neuro-retinitis.

Case 31. B. 372. Alfred H., $3\frac{1}{2}$; and his mother.

Failure of sight 2—4 weeks, after a severe illness of 2—3 weeks' duration, called "fever," (probably typhoid, for a sister had similar symptoms at same time, but her sight did not fail); vomiting, diarrhœa, headache, and emaciation; then rapid convalescence and failure of sight.

Neuritis, with pallor and swelling; symmetrical. Vessels of retina much diminished.

Ten *years* later (æt. 14). *February*, 1876. Has had excellent health, and been free from headache ever since the illness. Just sees 16 J.; sight has slowly improved since last attendance. Discs very white, but vessels not extremely diminished; edges considerably notched, and not quite clear; vessels moderately diminished. Clear evidence of past neuritis. No change since last seen at æt. 8 and æt. 4. Well grown and fairly intelligent. Still subject to occasional bad headache. Forehead rather square.

His mother is now found to have atrophy of her R. disc; it has been imperfect since she was 24. It shows no evidence of neuritis, and perhaps failed in connection with nervous headaches.

The eye diverges. V. 20 J, but field of V. much contracted. *o.d.* gray-white, clean and even; no signs of past neuritis. Vessels little, if at all diminished.

Believes it failed quickly in connexion with a severe inflammatory attack ("cold") before marriage. The eye was painful and bloodshot, and she was cupped for it. ? Erysipelas.

No other family history of bad sight.

Case 32. O. H. R., v, 311 (and B. 178), Case 4. Robert L., 1 year.

Neuritis (hazy discs and small arteries), with apparently complete blindness.

Supposed to have been blind some months, since having "fits" with teething. Ps. moderate size, and dilate well with atropine.

Discs subsequently cleared, and were not left very pale; arteries remained small.

V. improved decidedly during nearly a year.

No evidence of hereditary syphilis.

Case 33. O. H. R., v, 310 (and B. 173), Case 3. Louisa Butler, 12.

Neuritis, with woolly swollen discs, and total blindness. Blindness permanent (notes 2—3 years later).

History. Severe illness, with great pain in head, vomiting and delirium, loss of power over sphincters, and weakness of legs. Complete recovery in about 6 weeks.

Rapid failure of V. towards end of recovery from illness (*i.e.*, about 6 weeks after illness began).

Ps. wide and fixed. Paresis of R., int. rectus.

Probably a case in which neuritis did not cause damage to V. until it had been present for some weeks.

No note of the treatment adopted in the illness.

Case 34. B. 3. Esther E., 8. September, 1861.

Ten months ago had "gastric fever" for 1 month. Not very ill; no vomiting, and little headache. Dull and stupid, not delirious.

At end of month sudden loss of sight. Had had diplopia and squinting shortly before, and eyes said to have been very prominent.

Blue-white atrophy, and much diminished vessels.

Ps. normal size; act very little.

Is pale.

Case 35. F. 291. George M., 3½.

Amaurosis, with atrophy after neuritis (still passing off), after a cerebral illness; vomiting, severe headache, constipation, and feverishness; is said to have squinted. Was never so ill as to be kept in bed. Never became unconscious. No fits. Did not lose appetite. Has for long had a cough, and been "weak at the chest."

Ps. fairly active; eyes oscillate slightly.

A year later (æt. 4½) Ps. still act, but sluggishly, and are habitually large. Still quite blind.

Case 36. F. 186. Annie E., 14.

Blue-white atrophy of discs, with shelving cups, but little or no diminution of vessels. No p. l.

Ps. large and fixed.

Blind since "brain-fever," with very severe headache, and great prostration at æt. 4.

Now in excellent health.

Case 37. F. 92, and O. H. R. VIII, 493. Jas. Thos. Acton, 9.

Atrophy, after neuritis following cerebral symptoms, with great defect of sight.

Condition, 4½ years after the neuritis.

History of similar illness, ending fatally, in a brother.

Case 38. F. 68. Wm. Y., 14.

Advanced atrophy of discs, with evidence of past neuritis, viz., irregularity of choroid, and remains of lymph near disc. Arteries somewhat diminished. Refraction, Hy.

V., R. — cannot fix.

„ L. 12 J.

Failure of V. after a fit about 5 years ago; had convulsion of R. side of body.

Æt. 5 was operated on for convergent strabismus and for stone in the bladder by Mr. Hutchinson.

Case 39. G. 385. Thos. T., 27.

Atrophy of discs, without much diminution of retinal vessels; absolute blindness.

V. began to fail gradually during dropsy, after "scarlet fever," at æt. 10 or 12. The R. failed before the L. ? Was it albuminuric neuro-retinitis.

It was accompanied by very severe headache for some weeks. The head symptoms began the day after a hot bath during the dropsy.

The illness is said to have been without rash or bad throat, but was called scarlet fever by the doctor.

Case 40. G. 385. Jacob G., 24.

Atrophy of discs, with probable evidences of former neuritis; great defect of V. and constant oscillation.

Defect first noticed at æt. $3\frac{1}{2}$, when it was as now; this was immediately after whooping-cough at æt. 3.

There were no fits.

Strong and healthy ever since, and never subject to headaches.

Case 41. *Probably a case of Tubercular Meningitis with Neuritis, after Whooping-Cough.*

G. 320. Edward Golding, 3.

Failure of V. during or soon after whooping-cough. Was very ill with the whooping-cough, and so weak he could not stand for several months; bad pain at back of head; "one doctor thought water on the brain."

Gray-white atrophy of discs, without any positive evidence of neuritis. Vessels of good size. R. believed to be much more defective than L.

Doubtful choroidal changes near *o.ds.*

No alteration of manner or intelligence.

Subsequent doubtful improvement of L.

Enlarged glands under R. jaw.

Case 42. G. 271. F. H. C. S., $1\frac{3}{4}$ years.

Neuritis, followed by partial atrophy; temporary failure of V. for several months, followed by recovery.

The loss of V. accompanied a cerebral illness ("bilious attack"), with loss of power of walking, but with no enlargement of head. The illness began suddenly.

He had previously learned to walk well.

Some months after regaining his sight, he had again just managed to learn to walk.

Case 43. G. 70. Mary Anne A., 11.

Atrophy of discs after neuritis, and in R., near *y.s.*, some small spots of atrophy of choroid.

Amaurosis, nearly complete.

Failure of V. 2 years ago, with sickness and headache, after measles.

Case 44. C. 357. Henry M., 9.

Neuritis going on to atrophy (dirty-gray discs), and great defect (20 J.) coming on gradually during several months, and sooner in R. than L.

Failure, accompanied by convergent strabismus and diplopia.

Headache and retching during several months before admission. Never confined to bed.

Under care 9 months, and the strabismus passed off.

Mother probably remotely syphilitic, and has lost many children.

Case 45. C. 349. Hy. B..

"Cerebral fever," with pain in head and loss of use of limbs, followed by failure of V. æt. 8. Laid up a long time. Could not walk for some time.

Æt. 12. Well-marked atrophy of discs; V. $6\frac{1}{2}$ Sn. barely. Ps. not dilated.

Case 46. C. 296. Chas. B., 10.

Double neuritis (swollen woolly discs) and complete blindness; sight failing for 8 weeks.

Failure preceded and accompanied by severe frontal and circumorbital headache, and by attacks of pain in neck and spine as far as lumbar region. Pain coming on in attacks every two or three days, and accompanied by severe vomiting. Neck stiff.

? Cerebro-spinal meningitis.

Case 47. O. H. R., VI, p. 43. Ellen H., 11.

Neuritis following cerebral symptoms; convulsions, headache, ptosis.

Recovery of good health; 6 weeks later, blindness, with woolly discs.

Case 48. C. 73. Charlotte T., 7.

Atrophy after neuritis coming on during severe cerebro-spinal symptoms.

Blindness remaining 8 months later (perception of light).

Case 49. C. 19, O. H. R., V. 313. Case 8. Wm. F.

Neuritis passing into atrophy; amaurosis; no p.l. Ps. motionless, and R. much larger than L.

Coming on during a severe illness with fits and severe pain in head, 2—3 months before admission.

Weakness or inability to use the legs for a short time after the illness, but subsequent entire recovery of power.

Case 50. C. 14. Chas. H., $11\frac{1}{2}$.*

Neuritis, with an illness accompanied by paraplegia and insensibility. Blindness up to time of his death, 8 months after symptoms began.

Case 51. C. 16. Chas. S. W., 11.

Neuritis leading to blindness (permanent during the 6 months of his attendance), with pain in back of neck, but no head-symptoms.

Small-pox 3 months before sight failed.

Case 52. C. 35. Wm. Hy. F., 7.

Blindness, with atrophy (from neuritis), 4 months after failure of sight. Previous headache, but not severe. History of severe illness, "sore-throat fever," 8 months before admission.

* The three following have been published in vol. v, p. 309, &c., of these reports.

(*To be continued.*)

MISCELLANEOUS CASES.

By James E. Adams, F.R.C.S.,

Surgeon to the London Hospital, Assistant-Surgeon to the Moorfields Hospital.

Case I.—*Unusual form of Inflammation of the Eye occurring in a Gouty Patient.*

George B., æt. 53. Has frequently had rheumatic pains in his limbs, and has had five or six attacks of real gout, always affecting one or other great toe. Father and grandfather had gout—also a brother 10 years younger than himself. He is otherwise in good health, and does not drink. The following attacks have all been in the right eye, were all exactly alike, and passed away leaving no trace behind. They came on quite suddenly with great pain; the lids became œdematous (looking like an ordinary albuminuric œdema), and the ocular conjunctiva presented well-marked chemosis and slight redness. There was never a trace of iritis, his media remained clear, and V. normal. There was no evidence of sclerotitis as the red conjunctival vessels could be freely moved over the subjacent sclera which appeared to be normal in colour. The following are the dates of his attacks:—

No. 1, May, 1869. Treatment—Leeches; poppy fomentations; colchicum. Only attended once.

No. 2, January, 1870. Same treatment. Only attended once.

No. 3, September, 1870. Same treatment. Only attended once.

No. 4, March, 1871. Same treatment. Only attended once.

No. 5, February and March, 1873. Same treatment. Attended twice.

No. 6, November, 1873. Same treatment. Attended twice; much better on the 3rd day after taking medicine.

No. 7, November, 1875. Same treatment. Attended three times.

No. 8, November, 1876. Same treatment. This attack lasted rather longer than the others, and he attended 4 times, and he has not since presented himself.

Case II.—*Paralysis of Left Third Nerve with remarkable Intermission, Relapse, and Recovery.*

On June 11th, 1876, a gentleman, æt. 32, in large practice as a dentist, consulted me, complaining of slight ptosis in the left eye, with diplopia. At this time there was no *visible* deviation of the globe, but a chart* showing the position of his false image, revealed crossed diplopia (or divergent strabismus) with the vertical meridian of the left eye inclined outwards, or rather remaining too vertical in the movements upwards and to right, inclined a little out in the movement straight up, and corresponding to the right eye in movements to the left. On a level with the horizontal meridian and below it, all objects were seen single. This diplopia could only be referred to paresis of the left superior rectus. By the 14th the ptosis was more complete, the deviation of the left was visible, and the double images much further apart, and in a few days more the paralysis of the third was complete, excepting those branches to the ciliary muscle and iris. V. = $\frac{20}{20}$ and Ji in each. H.m. $\frac{1}{50}$.

There was a history of syphilis 10 years previously, apparently a slight attack. *Diagnosis.*—Gummatous tumour. *Treatment.*—Iodide of potassium in large doses, galvanism, and cold douche to lids.

At the end of a month he awoke one morning and found that he could open his eye, and that there was no longer any divergence or diplopia. This intermission lasted ten days, and then his symptoms gradually returned, reaching their former completeness in about 24 hours after the first recurrence of the diplopia. He became as bad as ever, and remained so for two months, when a gradual improvement commenced and went on to complete recovery. He is now (May, 1877) perfectly well, and attributes the improvement to the cold douche.

I can find no record of a similar train of symptoms in connection with paralysis of the third, or indeed of any other, nerve, and I would not have placed the case on record unless I were fully satisfied that the evidence was

* The chart should have been printed with the report of the case, but has been omitted in consequence of delay in going to press.

in all respects reliable; but here our patient was fully alive to the importance of every symptom, and accustomed to careful observation, and it is quite impossible to believe that such marked deviation of an eye could in any way be under the control of the will. To speculate on the possible causation of the intermission and relapse is idle, although a theory might be constructed to explain the unusual sequence of events.

A CASE OF GLYCOSURIC RETINITIS, WITH COMMENTS.

By Stephen Mackenzie, M.D.,

Assistant Physician to the London Hospital: Joint Lecturer on Pathology at the London Hospital Medical College.

The case I am about to communicate, illustrative of one of the ocular lesions associated with diabetes mellitus, may I hope prove of interest to ophthalmic surgeons. The case has a twofold value—clinical and pathological. It is valuable clinically, as diabetes was detected from ophthalmoscopic changes. It is valuable pathologically from the careful histological examination of the tissues of the eye by Mr. Nettleship; the cases being very few where a microscopic examination has been made, and but little being known as to the immediate cause of the retinal changes.

It may not be amiss if I preface the case by a brief review of what is known concerning glycosuric retinitis, as it will serve to direct attention to the most important points. In doing this I shall have to draw largely from Professor Leber's article, "Ueber die Erkrankungen des Auges bei Diabetes mellitus," which appeared in v. Graefe's Archives in 1875.* Professor Leber has collected eighteen cases of this disease, and added another, examined with great care and minuteness by himself.

Glycosuric retinitis rarely occurs until diabetes has been in existence for a rather prolonged period. It is generally ushered in by a more or less sudden and partial loss of sight, generally one eye suffers before the other, but in most cases the two eyes are eventually attacked, though often with unequal severity. Sometimes sparks are seen before the eye or eyes, at other times dark spots or muscæ are complained of, whilst in some cases disturb-

* V. Graefe's Archiv für Ophthalmologie, Band xxi, Abth. 3.

ance of the perception of colours is noticed. Pain does not appear to be of common occurrence. Patients usually have a succession of attacks of disturbed and limited vision, and between the attacks the sight may be in a great measure restored. In some cases the vision is almost completely lost in one or both eyes. On ophthalmoscopic examination the lens is usually observed to be quite transparent. The optic disc is sometimes found red, sometimes atrophic. The retina is generally cloudy; the vessels in many cases are distended and dilated, but in others, especially as regards the arteries, are smaller than natural. Scattered over the retina are apoplectic spots. These sometimes occur as round patches of various size, from large blots to punctiform ecchymoses; sometimes as linear streaks occurring in the course of the vessels; again at other times, as linear streaks running in a direction at right angles to the vessels. Sometimes the hæmorrhages are beneath the superficial layers of the retina, when the retinal vessels may be observed crossing the extravasation; at other times the hæmorrhages are superficial, and obscure the vessels. The hæmorrhages vary in tint according to their age. They often have a brownish-red hue. In addition to the hæmorrhages, white spots are seen scattered over the fundus. They are of various sizes and shapes. Sometimes they are of opaque whiteness, at other times they have a glistening appearance: those having the last character being generally smaller, and arranged in constellations. Such constellations are found grouped around the yellow spot, but are not limited to this situation. Occasionally longish white raised patches or folds, surmounted by retinal vessels, are seen. Some of the white patches are, doubtless, altered extravasations, for white patches are met with having red borders. Others would appear to be due to fatty degeneration of the retina. Though these appearances are seen with more or less clearness in most cases at some period of the disease, yet in other cases the details of the fundus are obscured by

opacities in the vitreous. Sometimes on examination nothing can be seen but a red reflex; sometimes floating films can be detected, having a red or brownish colour; at other times the vitreous is simply turbid and cloudy. These opacities of the vitreous are obviously due to hæmorrhages. They are found to occur coincidently with sudden limitations of the visual field, and they may be observed undergoing absorption.

The appearances in the retina are very like those met with in connection with Bright's disease. So close is the resemblance that some observers have passed them over as albuminuric changes, indicative of nephritis brought about by the diabetes. This opinion is favoured by the undoubted co-existence of albuminuria with glycosuria in the majority of the cases. Besides the presence of albumen, casts and blood have been found in the urine of some patients, who have, moreover, presented other evidences of Bright's disease, such as dropsy, hypertrophy of the heart, &c. Still, there remain a certain number of cases where it is expressly stated on competent authority, that there was no albuminuria. Thus in two cases recorded by Desmarres,* one by Noyes,† and one by Haltenhoff,‡ there was no albumen in the urine; in some of the cases the urine was examined repeatedly with care. In Noyes' case the amount of urea was on several occasions estimated and never found deficient. It thus seems clear that the changes are not necessarily of an albuminuric origin. In some of the cases the sugar had been noticed to almost or entirely disappear before the retinal affection was observed. What seems further to distinguish the intraocular changes in glycosuria from those met with in Bright's disease is the frequent complication of the former with

* Traité théor. et pract. des mal. des yeux. 2nd ed. Paris, 1858, t. iii, pp. 521—526.

† Trans. Amer. Ophth. Soc. 1869, pp. 71—75.

‡ Retinitis hemorrhagica bei Diabetes Mellitus. Zehend., Klin. Monats., 1873, xi, s. 291—298. An abstract of this and other cases will be found in "Periscope," parts II and III of preceding volume.

hæmorrhages into the vitreous. These occur equally in the cases where the diabetes is, and where it is not, complicated by albuminuria. Hæmorrhages into the vitreous are of rare occurrence in pure cases of albuminuric retinitis, so far as I know. They appear to be frequent in glycosuric retinitis, and are recurrent.

As to the cause of the changes in the retina and vitreous, there remains a certain degree of obscurity. Prof. Leber, in discussing these, considers that they may be due in part to the diabetes, in part to nephritis caused by the latter, or to the two combined. But, as he says, how diabetes brings about the changes is quite unexplained, as moreover, is the cause of the retinal affection in Bright's disease itself. But we find in by far the majority of instances of retinal disease that they are associated with lesions of other organs or with altered blood conditions and disturbances of nutrition. From the analogy of Bright's disease, in cases where the lesion is not due to renal affection, one is led to look to other organs in search of an explanation. The brain and the heart suggest themselves as possibly at fault. As regards the brain, the retinal changes met with in diabetes differ from the neuritis or neuro-retinitis usually associated with cerebral disease. Cases are met with, it is true, where changes almost precisely like those encountered in albuminuria are associated with coarse lesions of the brain, such as tumour, &c., but these are rare. Still, there may be some connection between the diabetes and the retinitis. Either the brain lesion may be primary, and cause both the diabetes and the retinal affection; or, on the other hand, the diabetes may be primary and cause both the retinal and the cerebral affections. These different classes of cases Prof. Leber has, he thinks, been able to distinguish in connection with the affections of the optic nerve in diabetes. Encephalic lesions, thus, do not appear to explain all the cases, and we turn to the heart in search of a cause. In the published cases, though the details in the records on

this point are few, there is nothing to support the idea that the hæmorrhages are connected with any heart affection, as in some forms of hæmorrhagic retinitis. In my case, about to be related, there was no hypertrophy or other sign of cardiac disease. Failing to find any adequate cause for the retinal changes in any of the viscera, we are driven, as Prof. Leber says, to compare diabetes with other morbid conditions attended by a like alteration in the retina, such as Bright's disease, pernicious anæmia, hæmophilia, purpura, leuchæmia, &c. In all of these diseases there is an undoubted alteration in the blood, and, in some at least, changes in the vessels, in consequence of which diapedesis of coloured corpuscles and transudation of abnormally constituted albuminous matters take place. Such alterations are not limited to the retina, but are generally found also in the brain, spinal cord, and many other parts of the body. Their wide distribution is evidence in favour of a common origin in a blood dyscrasia. Dr. Dickinson has discussed very ably* the relation between the changes he finds in the nervous centres, and which resemble the appearances seen in the retina and the glycosuria. I cannot help differing from his conclusions, however, for Dr. Dickinson regards the perivascular extravasations and erosions of nervous structures as the cause of the symptoms in diabetes. To my mind the whole evidence points to the changes in the nervous system as well as those in the retina and other organs, being due to an altered condition of the blood. If the retinal changes found in diabetes are not due to an antecedent brain lesion, but are dependent on the same cause as the latter, then the fact that glycosuric retinitis only occurs when diabetes has been some time in existence, becomes valuable as enabling us to correctly interpret the changes found in the nervous centres. From what we know of the other diseases where similar changes are found in the retina and

* Diseases of the Kidney. By W. Howship Dickinson, M.D. 1875.

in the nervous centres, such as pernicious anæmia,* hæmophilia,† purpura,‡ leuchæmia,§ ague,‖ the alterations in the nervous centres and in the eyes would appear to be coincident, rather than the latter sequential to the former. I think, therefore, we may justly infer, that the perivascular hæmorrhages, extrusions, excavations, and atrophies are the consequences and not the causes of the altered condition of the blood—I do not say the glychæmia—and blood vessels incidental to diabetes.

The following is the case to which I wish to direct attention :—

A woman (A. R.), æt. 52, admitted under my care into the London Hospital, December 15, 1875. Married; had neither children nor miscarriages. With the exception of an abscess of her knee-joint at 18 years, had no important illness; but had been subject to frequent attacks of diarrhœa, and suffered from rheumatic pains. The menopause occurred three years before admission. Her mother died of "galloping consumption;" her father through a severe cold. She had a brother living, but delicate.

She gave a rather confused and contradictory account of her illness, most of the facts having to be elicited by direct questions. In this way it was gathered that about three months before coming to the hospital, she went one day to look at the clock, and found she could not see what time it was with the left eye, and only with great difficulty with the right. This was the first time she experienced any defect of sight. Not being able to see the clock properly, she took a book in her hand and tried to read. She quite failed to do so with the left, and could not read at all plainly with the right eye. Shortly after this, as her sight

* Immerman, Deutsche Arch. f. klin. Med., 1874, xiii, 209–244. Manz. Veänderungen in der Retina bei anæmia progres. Centralblatt. f. d. med. Wissensch., 1875, No. 40, s. 675.

† Wickham Legg. Report from Post Mortem Room, St. Barth. Hosp. Rep., 1875, p. 64. Author. Med. Times and Gaz., March 10, 1877.

‡ Rec. Union Med., 1870, No. 48. Author. Med. Times and Gaz., March 3rd, 1877.

§ Poncet. Arch. de Physiol. norm. et Path., 1874, p. 496, and most ophth. writers.

‖ Author. Retinal Hæmorrhages and Melanæmia as Symptoms of Ague. Med. Times and Gaz., June 23, 1877.

did not improve, she came to the Ophthalmic Department of the London Hospital, and was seen by Mr. Waren Tay. Mr. Tay found hæmorrhages into each vitreous, obscuring the view of the fundus. In the left this was so much the case that nothing could be seen but an indistinct large white patch in the situation of the disc, with angular processes passing from it. In the right the margin of the disc could be made out, and did not show evidences of neuritis. Near the disc, below and to its outer side (inverted image) was a patch of extravasation of considerable size. Other smaller hæmorrhages could also be seen, but no white spots or patches. On examining the urine (when brought subsequently) Mr. Tay found marked evidence of the presence of sugar. The woman's health was failing, and her sight became so bad that she could not find her way about. Treatment of the diabetes afforded her only chance of improvement, and she was transferred to my care as an in-patient. At this time she could not read the largest test-type, and could not easily count fingers held in front of the eyes, but could distinguish the checks on the coverlet of her bed.

On inquiry it was elicited that about 12 months before admission she experienced severe pain in her back, especially in the region of the right kidney, accompanied by great thirst. Shortly after this she noticed that she passed a large quantity of urine, more than a couple of quarts a day. Her appetite was good, but not ravenous. In this state she continued up to admission, gradually losing flesh and strength, especially towards the latter part of this period.

On admission I noted that the patient was of short stature (4 feet $9\frac{1}{2}$ inches), fairly nourished, yet somewhat wasted; weight 6 stone $10\frac{1}{2}$ lbs. Her hair was plentiful, of a dark brown, turning grey, eyes greyish brown, complexion florid, lips of good colour; tongue small and pointed, coated with silvery fur on dorsum, but red at tip and edges; teeth few, those remaining in good state of preservation. There were no abnormal physical signs in chest, except a hyper-resonant percussion note and prolonged expiration, denoting vesicular emphysema. Owing to the latter, the cardiac dulness was masked. The heart sounds were natural. Pulse small, but rather resistent, the sphygmograph indicating a slight increase of arterial tension. Hepatic dulness in the right mammary line commenced at lower margin of sixth rib,

and did not extend beyond the margin of the costal arch. Splenic dulness not increased. Abdomen soft and flaccid. There was slight œdema of the feet. Her temperature was almost invariably below the normal, being generally between 97° and 98°. She passed on an average 3,000 cub. cent. of urine daily (rather over 100 ozs.), the maximum quantity being 4,000 c.c. The urine was pale and clear, and always had an acid reaction. Its specific gravity varied between 1,034 and 1,040. It contained on an average from 10 to 12 per cent. of sugar, giving about 380 grammes as the total quantity of sugar excreted per diem. The quantity of sugar varied somewhat, and rose (to about, on an average, 400 grammes) when she had been in the hospital a short time, with a liberal dietary. The largest quantity of sugar passed in one day was 666 grammes. She passed about 34 grammes of urea per diem, a quantity considerably in excess of the proper amount for a person of her weight. Her urine always contained *a trace of albumen*—never more than a trace. I have no mention of the presence of casts, which may be accepted as evidence that there were none, for the urine, I believe, was examined on several occasions microscopically. When the patient came under my observation the lenses were quite transparent, but the fundus was so obscured by the vitreous hæmorrhages that but few details could be seen. Mr. Couper saw the patient about this period, and his description accords with that of Mr. Tay, and with what I with less certainty made out. Mr. Couper reported: "In the *right eye* there are two large floating films in the vitreous; from their reddish-brown tint they are obviously altered blood clot. The vitreous itself is fairly translucent, yet turbid enough to conceal nearly all details of the fundus. There can, however, be seen an opaque, whitish, linear projection into the vitreous, on which retinal vessels are visible, and to which one of the filmy floating webs is attached. *Left eye:* the vitreous has all the appearance of having had diffuse hæmorrhage into it. It absorbs light strongly. The only visible detail of fundus is a glistening white crescentic mark, on which no vessels are visible, and which appears to be near the disc, but which cannot be identified with it. No visible floating material in vitreous. Tension fairly normal."

After being left on a mixed dietary for a week, her urine was tested hourly one day, for 14 consecutive hours, to ascertain the

period after ingestion at which the maximum amounts of sugar, urea, and water were excreted. It was found that the largest quantities of urine and sugar were passed at the third and fifth hours after a mixed meal, she having fasted 14 hours previously to this. At the third hour the quantity of urine was 250 c.c., and the sugar 31 grammes; at the fourth hour the urine fell to 80 c.c., and the sugar to 9 grammes; and at the fifth hour the urine rose to 200 c.c., and the sugar to 25 grammes. The urea did not follow so exactly the rise and fall of the urine and sugar, as it frequently does in diabetes; she passed about a gramme of urea an hour. At the eleventh hour after food there was a marked rise in the amount of urine, of sugar, and of urea, the latter reaching its maximum for the day—2·1 grammes in the hour.

On January 6th all starchy and saccharine substances were removed from her dietary, and she was ordered gluten bread, green vegetables, two eggs, four pints of milk, and three ounces of whisky per diem. A week later she said she felt better—not so sinking as she had previously felt; her thirst was less; she did not perspire; she had an acid taste in her mouth.

On January 31st, 1876, whilst still on an almost purely nitrogenous diet, she was ordered a grain and a half of carbolic acid, in a pill, three times a day; and on February 14th the dose was increased to two grains. During this period her general condition improved; her thirst was less; and she passed, if anything, rather less urine and sugar; the quantity of the latter daily excreted was about 250–260 grammes. Her weight increased from 6 stone 10½ lbs. to 7 stone 7½ lbs., which latter was reached at the end of February. From this time her weight decreased, and it will be seen that untoward symptoms made their appearance coincidently with this. There was no material change in the various constituents of the urine, which contained much sugar and a trace of albumen to the end.

Her appetite was never excessive; she never had diaphoresis. On February 17th she complained of shooting pains in her legs. On March 13th it was noted she had not been so well the last two days. Her face was flushed, though her temperature was slightly below normal. She slept badly; had fallen off in her appetite. She complained of "rheumatics" in her knees and ankles. On March 15th she felt weak and giddy, and could not stand. She vomited once; cared for no food. The carbolic acid was discon-

tinued, and a stomachic ordered. On March 22nd she drew the attention of Mr. Williamson, the house physician, to a twitching of the right leg, which she said had been going on for nearly a week. At my visit, the next day, she was lying in bed, with a very flushed face. The latter had a bright red congestive look, like a very rosy apple, and the redness was most marked over the natural prominences of the face, such as the nose, cheeks, and chin. Her tongue was coated with a whitish fur, and she complained of thirst. Her urine was noticed on this day to have a smoky tint, suggestive rather of carbolic acid poisoning than of hæmaturia. She had not taken any carbolic acid since the 15th, however, and this was the first occasion that the urine had presented this appearance. It had not a peculiar smell. There was a convulsive twitching of the muscles of the right leg and foot, by which the latter was extended and adducted by a succession of discontinuous spasms. She had pain in this leg and tenderness on pressure over the tibia. There was slight twitching of the left leg, but no pain or tenderness. There was slight pitting on pressure over both feet. Mr. Tay examined the eyes ophthalmoscopically on this day. He noted there was a "red reflex from all parts of the right fundus, except an undefined white patch in the region of the disc. The left fundus gives no reflex whatever—looks quite dark. Pupils (as usual, I may add) only moderately dilated." Up to the time of her taking to her bed, she had been able to make her way about the ward, more by the exercise of her senses of feeling and hearing than by sight. It is curious to contrast Mr. Tay's observation with the following statement of the patient on this day. She said she always felt as if there was a black hat over both eyes when she looks upwards, not when she looks forwards. When the left eye is closed she still sees the black hat, but when the right eye is closed the hat disappears, and she sees fairly well. She was ordered a mixture containing 15 grains of bromide of potassium and half a drachm of aromatic spirits of ammonia, in water, three times a day. Her condition remained stationary for the next few days; she had the flushed face and twitching of right leg.

On the 29th of March, Mr. Williamson was called to the patient at 6.30 a.m. The sister said that at 5 a.m. the patient became almost insensible very suddenly. A little whisky was given, but she continued to get worse, and when Mr. Williamson

saw her she was lying on her back, quite unconscious, with her eyes closed. When the eyelids were separated, the eyes were seen to be divergent; the corneæ were insensitive; the pupils were under the influence of atropine. Her breath had the odour of whisky. The pulse could not be felt at first, but a few minutes later it was just perceptible: it was then 120, regular, and extremely small. The respirations were 40 in the minute, and regular. They were deep and sighing; the chest rose with each inspiration, the mouth opening, and the head moving back. The redness of the cheeks, nose, and chin was less than during the past few days. The whole face had a dusky hue. The movement in the right leg had ceased. The surface of the body was moderately warm. She remained much in the same state until death, which took place at 7.30 p.m.

Autopsy: Body not much wasted. There was found a quite recent hæmorrhage, about the size of a wood nut, in the right *thalamus opticus*. No other noticeable changes in the central nervous system. The *lungs* were congested; they weighed 2 lbs. 3 ozs., and were free from pneumonic and phthisical changes. The *heart* weighed $7\frac{3}{4}$ ozs.; the left ventricle was moderately contracted; the valves and orifices were healthy. The *kidneys* were larger than natural, and weighed $13\frac{1}{2}$ ozs. The enlargement seemed to be due in great measure to increased thickness of the cortex, suggestive of some tubular disease. The *liver* weighed 3 lbs. 5 ozs.; the *spleen*, 4 ozs.; neither presented microscopic signs of disease.

Microscopical Examination.—In the *convolutions of the brain*, the principal changes are dilatations of the perivascular spaces. The spaces have irregular borders; in no case are they very large, not larger than three or four times the diameter of the contained vessel. Within the spaces, at parts, yellowish-brown granules are often encountered, generally in contact with the walls of the vessels. This pigment, from its colour, arrangement, and position, would appear to be altered colouring matter of the blood. Nowhere are seen unaltered coloured blood corpuscles outside the vessels, nor are blood crystals met with. Some of the larger veins are distended with coloured and colourless blood corpuscles, and surrounding many of the vessels are numerous small round cells, with one or two nuclei, which stain deeply with hæmatoxylin—colourless blood corpuscles—which have wandered from the

vessels. The arteries are for the most part healthy; the muscular coat is not thickened, nor are the nuclei proliferating. Only in a few places are noticed the hyaline changes of the intima, to be described more fully in connection with the vessels of kidney and spleen. The capillaries present no alterations. The periganglionic spaces are almost uniformly dilated.

Medulla Oblongata: Here the changes are very like those described in the convolutions, only the hyaline change in the arteries is more pronounced. The hyaline degeneration is mainly confined to the elastic portion of the intima. The endothelium presents no changes, but is sometimes separated from internal elastic coat. The hyaline change does not as a rule invade the middle coat of the vessels. The periganglionic spaces are much dilated; the ganglion cells being sometimes shrunk, sometimes retaining their normal size. The ganglion cells, especially when shrunk, are in parts deeply pigmented, of an almost mahogany-brown colour, and their outline is indistinct.

Pons Varolii: Same changes as in medulla; the perivascular infiltration with lymphoid cells is greater, however. Veins are met with filled with colourless corpuscles; but from there being no bulging of the walls or signs of stasis, this would not appear to be due to thrombosis. Some of the capillaries are markedly dilated, but for the most part uniformly so; only in a few places can any unequal dilatation of these vessels be seen, such as would deserve the title of capillary aneurism.

Kidney: Several important changes are met with here, but I will describe these with as great brevity as possible. First, as regards the uriniferous tubes. A noticeable feature is a dilated condition of the tubes almost throughout the whole of their varying course. This is well seen, for the membrana propria stands out conspicuously, the cells often being detached from it. In some parts the membrana propria is thickened by hyaline change. For the most part the secreting epithelium is well preserved, but in parts it is granular and indistinct. In the large collecting tubes of papillary portion, the epithelium presents very peculiar changes. In some of these tubes it has a beautiful, perfectly translucent appearance. The cells are swollen, and their outline is as distinctly marked out as in a nitrate of silver preparation. The cells are so translucent that there appears nothing to hold the nuclei in position. The nuclei, in some of these

altered epithelial cells, are of natural size, and well stained by hæmatoxylin; in other cells they are swollen to three or four times their natural size, and are pale and vesicular. They present all transitions between these two states, and the advanced stage reminds one of the swollen vesicular condition of coloured blood corpuscles when treated with water. The epithelial cells suggest the idea of having had all but the skeletons of the cells washed away, and there can, I think, be little doubt that the appearances I have described in the epithelium of the collecting tubes are due to the constant irrigation to which they were exposed. In other collecting tubes the epithelium is detached, and the lumen contains granular matter. Probably this is a later condition of the former change. Some of the tubules contain hyaline or fibrinous casts. There is a small amount of lymphoid infiltration at the bases of the pyramids. In the arteries a very conspicuous change is met with. It is most pronounced in the smaller arteries—the glomerular (afferent) arteries. These vessels are enlarged in an irregular way, and their walls greatly thickened. The increased thickness of the walls is mainly limited to the intima of the vessels, and consists of a hyaline transformation of this coat. The endothelium is, for the most part, intact, not swollen or altered in appearance. Sometimes it is crumpled up into a fold in the lumen of the vessel, and sometimes, whilst the individual cells preserve their mutual cohesion, it is detached from the rest of the intima. Between the endothelium and the muscle layer is a hyaline, yellowish-brown material, closely resembling in appearance the hyaline or fibrinous casts in the tubes, whilst their change involves the whole of this portion of the intima. Whilst this change involves the whole of this portion of the intima, it is rarely evenly distributed, but forms excentric bulgings at different points. The bulging is almost always towards the lumen of the vessel, which is in consequence greatly narrowed, or even obliterated. On transverse section of these vessels the lumen is often represented by an irregular space with crenated margin, the nuclei being brought very close to each other. When excessive, however, it bulges also outwards, so as, in a longitudinal section, to form a fusiform or biconvex enlargement of the wall of the vessel. Though usually confined to the intima, in some cases it involves the muscular coat, the purple stained nuclei of which may be recognised in its midst. The change usually commences just

where the afferent arteries are given off from the trunk of the interlobular arteries, the walls of the latter up to the very point where the altered afferent arteries leave them being generally unaffected. Sometimes, however, the interlobular arteries are markedly affected by the hyaline change, and here the bulging has as great a tendency outwards as towards the lumen of the vessel, appearing on longitudinal section as a plano-convex swelling. Here, too, the swelling is partial, different portions of the same vessels being affected in unvarying degrees. The hyaline change accompanies the glomerular (afferent) arteries throughout the whole of their course, generally ceasing at the point within the Malphigian corpuscles, where the vessel breaks up into capillaries. In the majority of cases the glomeruli are scarcely, if at all, affected; sometimes slight changes, similar to those in the arteries, are seen in the capillary network, rendering this indistinct. In some few instances the glomeruli have undergone atrophy, being reduced to a more or less fibrous body, in which are scattered a few nuclei. The latter change is similar to what is seen in a late state of interstitial nephritis. Sometimes, however, there is a slight increase of the glomerular nuclei. The muscularis is not thickened, unless it shares in the hyaline transformation. There is no increase in the thickness of the adventitia. The veins present no noteworthy changes. The capillaries are usually widely dilated, though for the most part empty. There are numerous deposits of amorphous matter staining purple with hæmatoxylin scattered about the kidneys, probably lime salts.

Spleen: The most conspicuous change in this organ is the hyaline degeneration of the arteries. This is extremely general, and very pronounced. It is confined to the arteries, especially the smaller ones. Many of these are completely obliterated along a considerable portion of their track by the walls being brought into apposition. Though the walls meet, they nowhere cohere. As in the kidney, the change is generally confined to the intima, though, in some instances, it also involves the muscularis. The endothelium is unaffected by the change, though sometimes stripped from the swollen elastic membrane. The swelling is irregular and excentric, in transverse section often appearing nodular. The hyaline material is almost of the same colour as that met with in the kidney; if anything, more yellow and refractive. Here, however, it has a tendency to separate by

cleavage into fibrillæ or lamellæ. I cannot decide whether this cleavage is determined by the position of nuclei. The adenoid sheaths of the arteries and the Malpighian corpuscles are somewhat enlarged, and present slight hyaline degeneration at the part immediately surrounding the altered arteries. *Liver:* There is atrophy of the liver cells which are found in various stages of fatty, granular, and pigmentary degeneration. The vascular and lymphatic capillaries are generally dilated. Some of the smaller branches of the hepatic artery show slight hyaline transformation of their intima. *Pancreas:* Epithelium more granular and indistinct than natural. Some deposits of amorphous and crystalline matter, taking a purple stain with hæmatoxylin—lime salts probably. Hyaline changes in the intima of some small arteries. *Heart:* Muscles fairly good; no important changes.

The posterior halves of the *eyes* were carefully removed. Large hæmorrhages in the vitreous were seen with the naked eye and the retinæ appeared thickened. After examination the globes were placed in preserving fluid and handed to Mr. Nettleship, who reports as follows:—

Microscopical Examination of the Eyes by Mr. Nettleship.

The principal changes discovered may be conveniently grouped into those in (*a*) the retinal and choroidal membranes and the vitreous; (*b*) the subvaginal lymph-space of the optic nerve and in the nerve itself; (*c*) and lastly, in the arteries and capillaries of the nerve, retina, and other parts.

(*a*) The *retina* was thickened in all its layers, the change being greatest in the nerve-fibre layer where there were numerous varicose swellings; the disc slightly swollen and its nerve-fibres converted into an intricate meshwork of fine threads, the meshes being for the most part empty, but sometimes filled by swellings like those in the retina. The thickening of the other retinal layers appears to be the result of chronic œdema with hypertrophy of the connective framework. The ganglion cells and the granule layers are well preserved, and indeed more readily seen than usual, their elements having been, as it were, dissected apart by the effusion of fluid between them; the fibres proceeding from the ganglion cells beautifully seen. Bacillar layer wanting and its place in some parts occupied by a layer of albuminous effusion. In the intergranule and molecular layers are here and there

round spaces (microscopic cysts) filled with large faintly-refracting globules (albuminous). *Limitans interna* much thickened in parts, and thrown into abrupt folds, which, when deep, resemble papillæ. The retina was much more thickened in the left than in the right; in the former it measured from two to three times its normal thickness. There were no distinct hæmorrhages in the retina, only one or two small patches of rusty-looking granules being seen near the disc.

The *vitreous* in the left eye contained a large quantity of blood, apparently of different dates, and forming a dense layer lying upon the *limitans interna* of the retina. The more superficial (vitreous) part often shows distinct lamination, the lines of which are sometimes arranged so as to suggest that the bleeding took place from different spots in a gradual manner, one mass of blood thus being intruded into another. The deepest (retinal) part of the clot contains many large dark rust-coloured nucleated corpuscles (resembling the cells of the vitreous) and granules (degenerated blood). Some of these coloured corpuscles and granules lie between the layers into which the thickened *limitans interna* is often split up. The source of the blood could not be traced.

The *choroid* at the margin of the optic disc is unaltered, but immediately on leaving that line it begins to thicken. The thickening increases rather rapidly, until at a distance of about one and a half or two discs' breadths from the border it is five or six times greater than normal; after this point it remains about the same. The change is greatest in the deep vascular layer, least in the chorio-capillaris, and intermediate in degree in the supra-choroidal space. It is caused (1) by great distension of the large vessels, both arteries and veins; and (2) by the collection of faintly-granular albuminous substance in the stroma between the vessels and in the elongated spaces of the *membrane supra-choroidea*. There is slight and doubtful increase of the colourless lymphoid corpuscles of the choroid.

$\frac{a}{b}$ (*b*) The trabecular tissue of the *subvaginal lymph-space* of the optic nerve is much increased in bulk, partly by thickening of the individual cords, partly by increase in their number. Excepting at the extreme ocular end of this space, there did not appear to be any increase of the cells clothing the trabeculæ, and even there this change was not great. The inner sheath of the nerve was somewhat thickened.

The *optic nerve* showed no very marked alterations. Its connective-tissue nuclei were increased in number in and just behind (= on the brain-side of) the *lamina cribrosa;* and further back (as far as three-fourths of an inch from the eye) they were unusually conspicuous, apparently from being somewhat swollen. The nerve-fibres were unusually varicose, and as the eyes were removed shortly after death in cold weather, it is probable that this was not altogether a *post-mortem* change.

(*c*) The most interesting changes are those in the blood-vessels, and although these are essentially similar to the changes in the vessels of the other organs examined by Dr. Mackenzie, a description of them will be of interest from the ophthalmological point of view. I must add that I did not fully observe and appreciate the vascular disease in the eyes until Dr. Mackenzie had shown and explained to me his preparations from the spleen, kidney, and other parts.

It is to be noted first, that there was great distension of all the vessels in the choroid and retina, as well as of the branches of the ciliary arteries surrounding the optic nerve in the sclerotic and of the nutrient arteries of the optic nerve-sheath.

The special changes in the arterial coats were, however, much more marked in the *arteria centralis* and its branches than in the scleral vessels, and were scarcely present at all in those of the choroid. The best marked alteration consists in a thickening of the layer immediately external to the endothelium, and this may occur either uniformly for a long distance or in circumscribed portions. Thus in the trunk of the *arteria centralis* there is a long tract of thickening between the endothelium and the fenestrated membrane, affecting, however, only one side of the vessel (as seen in longitudinal section) and rather suddenly increasing at one part so as to form a considerable swelling by which the lumen is notably contracted (Fig. 1); (a second and quite similar swelling is present lower down in the same vessel). In the retinal branches of this artery the thickening is much more general, although varying greatly in degree in different branches. As a rule the relative thickening of the coats is much greater in the very minute arteries than in those of larger size (Figs. 2, 3, and 4). In an early period the new material is more or less distinctly corpuscular (Figs. 1 and 2), and its innermost portion sometimes appears composed of superimposed layers of endothelia

(Fig. 1). In many specimens, where the disease is apparently of older date, the appearance is best described as partly hyaline and partly fibrous (Figs. 3 and 4), the hyaline change being most

Fig. 1.

Fig. 2.

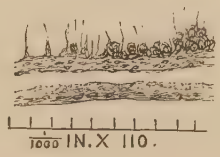

Fig. 3.

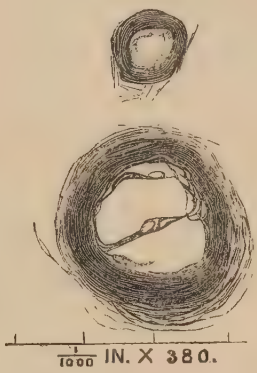

Fig. 4.

Fig. 1. *Art. Centr. Retinæ.* Longitudinal section. Upper end of drawing is at level of deepest part of *lam. crib.* Thickening of one side of vessel, and narrowing of lumen from nucleated growth between endothelium and elastic lamina; detachment of endothelium; (?) increase of muscle cells opposite thickest part of swelling.

Fig. 2. A rather small artery from nerve fibre layer of retina. Müller's fibres and ganglion-cells bound one margin (the upper in the figure). Thickening of the entire arterial wall, with separation and some obscuration of nuclei (this point not well shown in the woodcut).

Figs. 3 *and* 4. A minute and a medium-sized retinal artery seen in transverse section. Hyaline thickening of coats with indistinctness of the muscle nuclei; in the large vessel a more fibrous appearance. Alteration of the endothelium.

obvious in the smallest vessels. The endothelium is often separated in single cells or in irregular sheets (Figs. 1 and 4), and in the smallest vessels sometimes looks as if swollen (Fig. 3). The

relation of the muscular coat to the morbid change is not easy to make out in all cases. In such a vessel as is depicted in Fig. 2 it appears that the whole vascular wall external to the endothelium is swollen, but on examination with high power no evidence of increase in the muscle-cells is apparent; rather the whole tissue appears swollen and dim, the nuclei being less apparent than in health (the nuclei as represented in the woodcut are too conspicuous). In others (as seen in Fig. 1) there is some increase of corpuscular elements in the situation of the muscular layer just at the part where the greatest internal thickening is present. In the more hyaline vessels (Figs. 3 and 4) it is difficult to make out any trace of the muscle layer, only a few obscure nuclei being visible. A uniform thickening of the fenestrated membrane is the only change visible in a tolerable number of arteries.

The veins as a rule are merely distended, their coats not altered. Some of the large branches on the disc, however, pre-

Fig. 5.

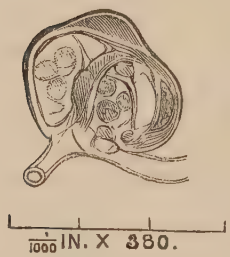

Fig. 6.

Figs. 5 and 6. Two retinal capillary aneurisms. One contains a mass of brownish-red granules of various sizes. The other is (or appears to be) chambered by septa or bands, probably altered endothelia; some globules of albuminous matter and altered blood corpuscles seen in its cavities.

sent numerous cells like leucocytes, in and immediately outside their coats; this, which is a perfectly definite change when it occurs, is noticed in only a few of the veins. It may here be noted that some of the blood-vessels in the tissue outside the sclerotic were surrounded by circular thin-walled spaces, probably distended lymphatic sheaths.

The capillaries of all parts are distended. In the retina there are minute aneurisms on the capillaries. These are not very abundant; they often occur in little groups of about half a dozen or less near together, large adjoining tracts being quite free from them. They are globular pouches, apparently communicating with only one side of the vessel; never fusiform dilatations (Figs. 5 and 6). They usually contain some reddish-brown granules, and sometimes similar granules are seen lying in the tissue just external to them, suggesting that the bleeding into the vitreous may have been due to the consecutive rupture of a number of these minute sacs. One of them appears to be divided into chambers (Fig. 6). The capillary walls are as a rule not much, if at all, thickened, but here and there considerable thickening is seen.

It will be seen that the eye-disease first manifested itself in a characteristic manner by disturbance of vision; and that polydipsia and polyuria had preceded the ocular symptoms for about a year. The diabetes was severe, as evidenced by the amount of urine and sugar daily excreted. The patient generally passed about a pound of sugar per diem, and sometimes considerably more. Further, the patient died, whilst her blood was loaded with sugar, with that peculiar form of coma which has been termed "diabetic coma," or "coma-collapse," and which has been attributed by Kussmaul* and Rupstein† to the development of acetone in the blood—*acetonæmia*.

It may be thought that death was determined by cerebral hæmorrhage, for there was an extravasation into the

* Zur Lehre vom Diabetes Mellitus, Deutsch. Arch. f. klin. Med., 1874, xiv, 1–47.

† Centralblatt f. die Med. Wissensch., 1874, No. 55, s. 865.

right thalamus; but I do not think this had necessarily or exclusively to do with the coma. I have watched several other patients die in quite a similar manner, and on *post-mortem* examination, no gross change, such as hæmorrhage, has been found. This is also the general experience. The hæmorrhage found was recent, and was on the same side as the convulsive movements noticed in the right foot. It could not, therefore, have given rise to the latter, though the cause of these movements was not discovered. The cord has not been carefully examined microscopically. It will be observed that the patient's urine contained albumen, and it becomes of importance to try and determine what share, if any, the kidney had in producing the retinal changes and extravasations into the vitreous. It will be noticed that the urine contained only "a trace" of albumen throughout the observed progress of the case. It was daily examined. There was, also, slight œdema of the feet. The kidneys were larger than natural on *post-mortem* examination, and the increase was mainly of the cortex. The microscope shows slight changes in the tubes, and a very slight degree of intertubular infiltration. These changes undoubtedly indicate a very slight amount of parenchymatous and interstitial nephritis, and are quite in harmony with the persistent presence of a small quantity of albumen in the urine, and the œdema of the feet. But the renal affection was really very slight, and the kidneys were able to perform their functions, as evidenced by the amount of urea daily excreted by them. A further proof that the kidney disease was not severe or of long standing, or that the general vascular degeneration—"arterio-capillary fibrosis"—was non-existent, is that no hypertrophy of the heart was present. The amount of renal disease appears no more than their overtaxed functions would be likely to induce. The increased bulk of the kidneys (the two weighing 13½ ounces), was mainly due to the dilated condition of the uriniferous tubes and blood-vessels. There was a peculiar change in

the arteries of the kidney, but this is distinct from the changes usually met with in Bright's disease. There, therefore, appears no clear evidence that there was such an amount of renal disease as to call forth secondary changes, like retinitis. For the same reason, there is nothing to warrant the opinion that the coma was due to uræmia. I think, therefore, we are justified in excluding the kidneys as the cause of the changes in the retina and vitreous. There was no disease of the heart to account for the hæmorrhages. As regards the minute changes in the intra-cranial organs, they are slight and similar to what have been noticed before,* with the exception of the hyaline change in the intima of the arteries. Similar vascular changes are also found by Mr. Nettleship in the retina, but are not peculiar to, or most marked in, nervous structures. With reference to this hyaline change of the intima of arteries, from my description it will be seen that, though widely distributed, it is most marked in the kidney and spleen, and its nature can be best studied in those organs. It seems to be a peculiar transformation of the elastic portion of the intima, though it sometimes, as Mr. Nettleship has noticed in the retina and optic nerve, and I have observed in the kidney and spleen, extends beyond these limits and invades the muscular coat. The material in question has a peculiar faint yellow to yellowish-brown colour in logwood preparations, is somewhat highly refracting, and appears homogeneous, with a tendency to become fibrillar. It is not acted on by iodine or methylaniline in a similar manner to amyloid or lardaceous material. This change is a very interesting and important one. It is identical with what Dr. E. Klein has described in the arteries of the kidney and spleen in scarlatina,† and in the spleen in enteric fever.‡ Fr. Neelson§

* Dickinson, loc. cit.

† Reports of the Medical Officer of the Privy Council. New Series, No. VIII.

‡ *Ibid.*, No. VI.

§ Archiv der Heilkunde, 1876. Heft ii und iii, s. 119.

has noticed a somewhat similar change in the capillaries of the pia mater in some of the acute infectious diseases and in various other diseases (syphilis, phthisis, pneumonia, dysentery, rheumatism, &c.), and other observers have noticed colloid or glassy degeneration of arteries and capillaries of the pia mater in connection with brain diseases. The change in my case exactly corresponds with what Dr. Klein has so well described and delineated, in connection with scarlatina and enteric fever, with what I have found (in the spleen) in all the cases of pyæmia I have examined, and with the appearances seen in a case of sub-maxillary cellulitis (angina Ludovici) I have investigated. I can quite corroborate Dr. Klein's statement that the change is external to, and independent of, the endothelial lining of the arteries, and that it is situated in the elastic membrane of the intima. The change, Neelson observed, is similar to the "hyaline fibroid" change of the adventitia, first described by Sir Wm. Gull and Dr. Sutton,* from which this change in the intima is distinct. I have found this change in a slighter degree in other cases of diabetes, but am not aware of its having attracted other observers' attention.

Though this hyaline transformation of the intima well deserves, from its effects, the title of "obliterative arteritis," it is quite different from the "obliterative arteritis" described by Friedländer,† or the change identical to the latter, described by Heubner,‡ under the name of "syphilitic degeneration of the cerebral arteries." The change in the intima of the renal arteries, in my case, is greater than in any case of scarlet fever I have seen, or in cases of pyæmia. It is interesting to note that in most of the diseases it is met with, there is some profound alteration in the blood; and this altered condition of the blood, and of relation between blood and blood-vessels, may

* Medico. Chirurg. Trans. 1872, p. 273.

† Centralblatt f. d. med. Wissensch. 1876, No. 4, s. 65.

‡ "Ueber luetische Erkrankung der Gehirnarterien," Leipzig, 1875.

have something to do, also, with the hæmorrhages. It is noteworthy that Dr. Klein has met with the hyaline change of the arteries and capillaries most marked in early cases of fatal scarlatina, before there is any interstitial nephritis, and he finds no reason to believe there is any relation between the two. In most of his cases there was no albumen in the urine. He also finds in scarlet fever proliferation of the muscle nuclei in vessels presenting this change, though not limited to these. In my case, there is no increase in the muscle nuclei.

The occurrence of similar hyaline changes in the arteries of the retina (as illustrated in the woodcuts) is a point of much interest; although it must be remembered, that the changes are not so pronounced in the retina and optic nerve as in the kidney and spleen, more resembling those in the liver and pancreas. But a point of still greater importance is the existence of aneurisms of the retinal capillaries. Their existence may serve to explain, as Mr. Nettleship suggests, the hæmorrhages into the vitreous. Professor Leber is also inclined on other grounds to attribute the vitreous hæmorrhages to repeated bleedings from small retinal vessels.

CASES OF FOREIGN BODY IN THE EYE.

By Frederick Mason,

Surgeon to the Bath Eye Infirmary.

Case I.—*Foreign Body in Eye for seventeen years, ten years without producing active disturbance.*

H. W., æt. 32. Was admitted an in-patient of the Bath Eye Infirmary on the 26th February, 1870, for what was supposed to be a case of severe catarrho-rheumatic ophthalmia of the right eye, there being at that time no appearance or history of accident. There was chemosis, iris inflamed and pupil fixed. This inflammation had commenced seven weeks previously. He had been under treatment, had taken mercury, and the gums were spongy and bleeding.

On the 26th March the inflammation had subsided, the pupil elongated upwards and outwards, and not dilatable, the eye was quiet, and he was discharged from the infirmary.

On the 3rd March, 1877, he was readmitted.

Right not inflamed. Cornea less in diameter than left, with an indistinct scratch-like scar running across the lower half. Anterior chamber small. Iris tremulous, with appearances of former inflammation. Pupil central, round, and fixed. Sclerotic blue, in patches. T.n. No perception of light.

Left. Can read J. 2, but it becomes watery on looking at near objects. Os. od. normal. Choroid dark, and no reflection of red light from it.

The scar on right cornea giving rise to a suspicion that the eye had been injured by accident, he was closely questioned, when he recalled to remembrance the following circumstance which, not thinking of any importance, he had until now forgotten :—

In September, 1860, he was working for an ironmonger, and was engaged knocking iron hoops from a cask with an iron chisel when a piece flew off and struck the right eye. He says the piece came out of the eye on the following day. He suffered but slight inconvenience, and did not apply for advice for five or six weeks, when vision becoming dull he consulted the late Mr. Vicary of Warminster. The eye was then inflamed for two days.

Vision continued good, but not equal to that of the other eye, for 10 years, that is until the attack of inflammation in 1870. After leaving the infirmary in that year, he retained vision for 12 months, then quietly losing it without pain or any further inflammation. During the last two years the eye has been occasionally very painful and sometimes acutely inflamed; the inflammation lasting for a few hours only, and for about the same time the left has been watery and weak.

On March 4th, 1877, the right eye was enucleated. On making a transverse section through the sclerotic, dividing the globe into an anterior and posterior half, the vitreous was found to be of a very pale straw colour, very fluid, and of not greater density than the aqueous. Behind the retina and surrounding the od. lymph had been effused apparently in the course of the vessels. There was no trace of lens, but some shreds of capsule behind the iris. The pupil was filled with a semi-transparent lymph.

Firmly imbedded or encysted within the fibres of the iris a foreign body could be felt, not seen; this was with difficulty removed, and proved to be a minute fragment of iron, ⊙ which must have entered the eye at the time of the accident 17 years before, remaining in it for 10 years without causing any active disturbance.

On the 10th March, the wound being nearly healed and vision of the left much improved, he was sent home.

Mr. Willcox, of Warminster, has furnished me with the following additional particulars:—

June 5th 1877. "I have carefully examined H. W.'s eye this evening. There is no sign of any inflammatory condition. The cornea and all the media are clear; there is no congestion of the vessels of either the conjunctiva or sclerotic. He says the eye is somewhat weak, and there is slight lachrymation when exposed to the sun or the wind. He can readily decipher the addresses of letters (he is a letter carrier), and he can recognise people easily across the road. The pupil dilates readily and regularly with an atropine disc, and the ophthalmoscopic appearance of the fundus is normal. He volunteered the statement that he is entirely relieved of the brow pain, which before the operation had been very great."

CASE II.—*Foreign Body in one Eye for Four Years before Sympathetic Irritation of the other.*

J. W., æt. 62, was admitted an in-patient of the Bath Eye Infirmary, May 29th, 1877.

On the 5th November, 1873, when breaking stones, he was struck on the right eye and received a wound of the cornea; he became a patient at the United Hospital, and remained under treatment there for seven weeks. He has at various times sought advice at other institutions.

Within the last week vision has failed in the left; and everything now appearing cloudy and dull he came to the Eye Infirmary.

Right. Cornea opaque at the lower and outer border showing the scar of an old wound which penetrated through the iris and ciliary region; pupil obliterated. T −1. Vision nil, and he has frequent attacks of pain in the right. Left. Os. media hazy, od. not clearly seen, choroid dusky with numerous faint cloudy black patches. T + 2. Vision: perception of large objects, but everything hazy.

May 30th. Right enucleated. On making a section a piece of carboniferous limestone, flat and quadrangular in form, about $\frac{1}{5}''$ long, $\frac{1}{8}''$ wide, and $\frac{1}{16}''$ thick on one edge, but sharp on the other, chisel shaped, was found lying upon the posterior surface of the ciliary processes, between them and the remains of the lens capsule, and surrounded with organised lymph which was firmly adherent to the stone. The lens was absorbed and the retina detached and forming a fold of membrane stretching backwards from the capsule. Deposits of lymph on choroid.

June 2nd. Vision of left already much improved, cloudiness gone, he can see the hands and tell the time by the watch.

9th. Considers himself quite well, and vision as good as ever.

Os. media clear. The cloudy dark patches on choroid have disappeared. The intervascular spaces are of a dark colour, but well defined. Od. normal. He has never learnt to read, but can distinguish the spaces between the letters in J. 3.

CILIO-RETINAL BLOOD-VESSELS (ADDITIONAL CASES AND REMARKS).

By Edward Nettleship, F.R.C.S.,

Surgeon to the South London Ophthalmic Hospital.

Since recording elsewhere* some cases in which a communication between the retinal and ciliary vascular systems could be seen by the ophthalmoscope, I have been on the watch for other examples. The result has shown, as was suspected, that this condition is much more common than has been supposed,† so common indeed that when the attention has once been drawn to the subject, scarcely a week passes without one or more examples coming under notice in the ordinary routine of ophthalmoscopic work. I have not attempted to make notes of all the cases in which I have seen this deviation, but have appended to these remarks seven additional cases, some of them with diagrams, and have also reproduced the diagrams and descriptions of cases originally published in the British Medical Journal. This series of figures, although rudely executed and much simplified for economy by the omission of unessential details, may, I think, be taken as representing with sufficient accuracy some of the commonest, as well as the more exaggerated, types of this anatomical variation.

In the earlier observations I was obliged to infer that the errant retinal vessel ran into the choroid or sclerotic, never having been fortunate enough to prove in a microscopical specimen the presence of a vessel taking this course, and yet large enough to have been visible by means of the ophthalmoscope.

* British Medical Journal, Feb. 5, 1876, and Ophth. Hosp. Reports, vol. viii, p. 512.

† See Leber in Graefe & Saemisch's Handbuch d. Augenheilkunde, ii, 1, 307; and Loring, in Knapp's Archives II, ii, p. 9.

In examining lately the sections of a swollen (*neuritic*) disc from a case of cerebral tumour (Amelia Childs, No. 42), which had been under care at Moorfields, I found in one of them an artery of considerable size passing from the sclerotic at the level of the upper surface of the *lamina cribrosa* into the optic disc, where it could be followed nearly as far as the free (vitreous) surface (Fig. 1). The course taken by this vessel is such that it could not but be distributed to the retina. The section was made in the horizontal plane, and the vessel alluded to is on the side next the *y.s.*, as proved by the characters of the retina, &c.; the position of this vessel, therefore, agrees precisely with that of the similar vessels observed during life.

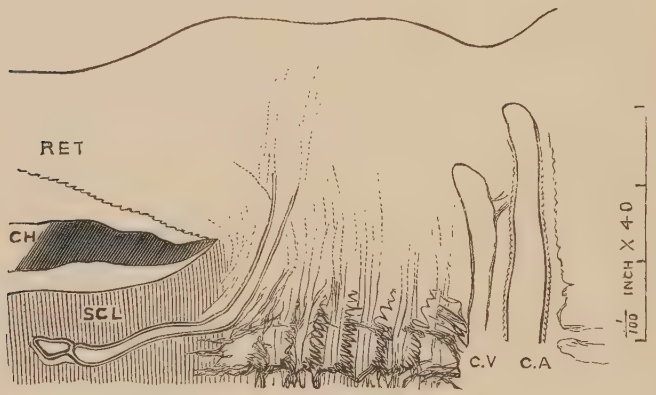

Fig. 1. Section showing a large artery passing from sclerotic into optic disc and retina on the *yellow spot* side. Its diameter is from $\frac{1}{3}$ to $\frac{1}{2}$ that of the *arteria centralis* (C.A.). C.V., *vena centralis*. × 40.

It may be suggested that in this case (Case V*) the communicating artery may have become somewhat enlarged to compensate for the diminished supply of blood delivered through the constricted scleral ring by the *arteria centralis*. Two considerations, however, render it unlikely that such an enlargement would be very considerable in a case of neuritis, such as this: (1) The sclero-retinal vessel is itself

* The numbering continued from the former series, vol. viii, p. 513.

subject to pressure at the scleral ring, only slightly less in degree than that exerted on the *arteria centralis;* (2) It is well known that the arteries in the retina do not anastomose, unless here and there by means of a few capillaries (see vol. viii, p. 262 of these Reports); and in all probability, therefore, no enlargement of a single arterial branch could add materially to the blood supply of any but its own proper segment of the retina.

The following general statements may be made on the subject of this deviation:—

1. It is common to meet with healthy eyes in which a retinal vessel on reaching the disc turns sharply back and disappears under the rim of the choroid, as if running into (or emerging from) either the choroid or sclerotic.

2. This cilio-retinal vessel may be either an artery or a vein. It is generally very small (see Fig. 8), but may be as large as one of the main divisions of the *arteria* or *vena centralis.* (Figs. 2, 3, 4.)

3. Such vessels are always situated on the temporal side of the disc, and pass to (or from) the yellow-spot region of the retina.

4. Such a vessel is generally present in only one eye, but is occasionally met with in both. It is usually single, but two vessels may take this course in the same eye.

5. A cilio-retinal vessel can in most cases be readily distinguished from the other branches (generally of small size), which are often seen to enter (or emerge from) the optic nerve at various points between the temporal border and the physiological pit. There is some difficulty in drawing this distinction when the vessel disappears very near to the border of the disc, and it may be impossible to say in such a case whether the vessel passes straight down the nerve or curves back to join the sclerotic.

6. Anatomists are not agreed* as to whether those vessels which disappear between the border and the physiological pit are branches of the central retinal vessels

* Leber, loc. cit.

given off before reaching the eyeball, or branches of the ciliary vessels from the sclerotic, and the fact that the latter connection can often be demonstrated during life for one vessel makes it highly probable that many of the other small branches alluded to are also offshoots of the ciliary system.

7. The cilio-retinal vessels do not appear to anastomose with the other retinal vessels either on the retina or optic disc.

8. Whenever the case admits of complete demonstration (Figs. 1, 4, and 6), the vessel joins (or emerges from) the sclerotic, not the choroid. It is at present uncertain whether it is ever a branch of a choroidal vessel.

9. The presence and size of this communicating vessel does not appear to bear any relation to disease. It is, when present, more easily recognised and traceable further, if there be an atrophic crescent, than when the choroid at the border of the disc is healthy. It often seems to taper off a little towards its point of submergence in the choroid or sclerotic; this appearance being explained in the same manner as the similar apparent tapering of the *arteria* and *vena centralis* in the physiological pit, and not indicating any diminution in size.

10. No practical significance can at present be attached to this peculiarity in the blood-supply of the yellow-spot region of the retina. Should further observation show that several of the smaller vessels passing directly from the disc to this region are generally branches of the ciliary vascular system, and not derived from the central retinal vessels, the fact may throw light on some of the functional and structural diseases of this important part.

Additional Cases.

Case VI.—Thomas S. (Mr. Hutchinson's patient for syphilitic retinitis; T. 123). In the left eye a large artery, distributed to the parts below the yellow-spot, emerges from under the choroidal rim of the disc, and after making a very short and abrupt curve,

passes on to the retina. (Fig. 2.) The border of the optic disc is even and unpigmented everywhere, excepting just where this artery emerges, but here it is notched slightly outwards, and the pigment appears to be somewhat heaped up, forming an abruptly black edge. (Fig. 2.)

Fig. 2 (see Case VI).

Case VII.—Walter S., æt. 18. Under care for disease of the vitreous without apparent cause, in the left eye. Refraction hypermetropic, but with some astigmatism; a narrow crescent of choroidal atrophy bounds the disc at its (real) lower and outer part. A retinal vein of considerable size, passing from below the yellow-spot region inwards and upwards, on reaching the surface of the disc curves back, leaves the disc, again crosses the surface of the atrophic crescent, and disappears under the border of the choroid. (Fig. 3.) This note was made after the vitreous had to a great extent cleared, vision with this eye being 1 J. fairly, and $\frac{20}{40}$ Sn.

Fig. 3 (see Case VII).

Case VIII.—James E., æt. 21. Myopia 9 *d.* in right, and 13 *d.* in left. In both a large abruptly limited posterior staphyloma, and in the left some separate patches of atrophy near the yellow spot; no changes at the periphery of the fundus.

In the left the staphyloma entirely surrounds the disc, being wider on the yellow-spot side. A retinal vein of the second magnitude passes from the (apparent) upper-inner part of the fundus (apparently above the yellow-spot region), makes an acute S bend and dips into the exposed sclerotic forming the background of the staphyloma at a distance from the border of the disc equal to about one quarter the diameter of the latter. (Fig. 4.) A large

choroidal vessel is seen, somewhat dimly, in near relation to this retinal vein on the staphyloma, but there is no proof of a junction between the two. The appearances, and particularly the abrupt bend made by the vessel, could be accounted for by supposing that before the staphyloma was formed the retinal vein passed on to the disc, formed a loop, and disappeared in the usual manner beneath the choroidal rim; but that in consequence of the stretching and displacement of parts in the formation of the staphyloma, the single loop was converted into an S quite removed from the disc, while the point of entrance of the vessel into the sclerotic became exposed.

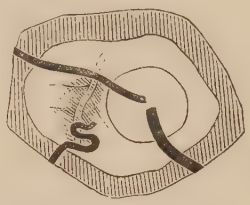

Fig. 4 (see Case VIII).

Case IX.—Eliza T., æt. 67. A patient with atrophy of the discs of many years' standing, probably following neuritis. Right eye no p.l.; left sees 19 J. barely, with + 5 = 12 J. barely.

In the left a vessel of about the fourth magnitude appears to join the sclerotic, as in the other cases, at the yellow-spot side of the disc. There is no crescent, however, and the vessel therefore cannot be actually seen running into the sclerotic. It was apparently an artery.

Case X.—Emily B., æt. 23. High myopia, with a staphyloma entirely surrounding the disc in each eye. In the right, which has more than $\frac{1}{3}$ of myopia, two retinal vessels join the sclerotic or choroid in the usual manner.

The following woodcuts (Fig. 8 excepted) are reproduced from the British Medical Journal, Feb. 5, 1876:—

Fig. 5.—Frederick M., æt. 12. Highly myopic, with a tolerably sharp, rather narrow staphylomatous crescent. A vein of the third magnitude passes from the (apparent) lower-inner part of the fundus (*i.e.*, really from above the *y.s.* region). On reaching the border of the disc, it turns back and disappears at the junction of the atrophic and healthy choroid. A small venous twig

coming directly from the *y.s.* region joins this vessel just as it is about to disappear.

Fig. 5.

Fig. 6.

Fig. 6.—Gertrude H., æt. 25. Myopia $\frac{1}{7}$, with a wide crescentic staphyloma on the *y.s.* side. A retinal vein of the fourth size passes inwards from the *y.s.* region, crosses the staphyloma, bends sharply upwards just before reaching the disc, and disappears partly upon and partly external to the scleral border. It appears to enlarge slightly at its point of disappearance.

Fig. 7.—Lizzie W., æt. 20. Hypermetropia. An artery of the third or fourth size passes from below the *y.s.* region on to the disc, curves back, and disappears at the choroidal rim or just external to it. (A minute twig, which was figured in the original case as appearing to connect this vessel with another on the disc, has here been left out, the evidence of anastomosis being imperfect.)

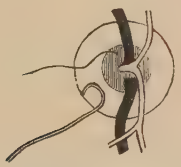

Fig. 7.

Fig. 8.

Fig. 8.—Ellen S., æt. 35 (Case II, vol. viii, p. 513).—Atrophied right optic disc, showing a small vein running from rather below the *y.s.* turning over the edge of the disc and disappearing under the choroidal rim. There had been no intraocular neuritis.

The vessel in this case presents a good average example of the variation described; in the majority of cases it is of about the size here shown.

CLINICAL NOTES AND CASES.

By Edward Nettleship, F.R.C.S.,
Surgeon to the South London Ophthalmic Hospital.

(Continued from vol. viii, p. 520.)

The cases given below illustrate the following diseases:—

1. *Retinitis pigmentosa*—four cases with family histories, two of them extending back in considerable detail for two or three generations. These cases, while strongly confirming the frequency of heredity in this disease, show no evidence that blood relationship in parents has any special influence. (Cases 1 to 4.)

One of the patients (Case 1) presented a particularly symmetrical, regular, and uncomplicated form of posterior polar cataract, of which it seemed worth while to give a woodcut.

There was a history of serious disease of the nervous system, in one or more previous generations, in three of the cases.

2. Two cases of choroiditis or choroido-retinitis, with history of diseases of the nervous system in brothers or sisters. One of the patients is herself the subject of peculiar nervous symptoms. Hereditary syphilis was not excluded in either of these cases. (Cases 5 and 6.)

3. A case of amaurosis with atrophy of discs preceding general paralysis of the insane. (Case 7.)

4. A case in which a man presenting many of the most marked symptoms of disseminated sclerosis (as described by M. Charcot in his Lectures translated by the New Sydenham Society) has been under care on account of his ocular symptoms for nearly three years. (Case 8.)

5. Localised exudative choroiditis, with retinitis, at or about the age of puberty, and without recognised cause. These cases are very peculiar, and deserve most detailed study from the etiological and pathological points

of view. In ophthalmoscopic appearance the diseased patch in such a case bears more resemblance to a tubercle in the choroid than to any of the ordinary forms of choroiditis. It becomes a question whether in strumous or tubercular subjects, such as these patients were, localised inflammation, approaching suppuration, may occur in the deep structures of the eye without evident cause. Whether strumous or not, it is important to enquire whether such changes as may be seen in the eye in these cases may take place in other important organs, especially in connection with the nervous system. (Cases 9 and 10.)

I believe that cases resembling the two here given are by no means common, but remember to have seen one or two closely similar to them in Mr. Hutchinson's practice at Moorfields. Illustrations of identical ophthalmoscopic changes are given in Dr. Hugo Magnus's Ophthalmoscopic Atlas, T.V., Figs. 1—4.

6. Examples of the "choroido-retinal disease," described by Mr. Hutchinson and Mr. Tay in a former number of these Reports (Vol. viii, p. 231). The first case is of especial interest, because definite though incomplete atrophy of the discs has followed the condition of slight haziness which, in the early period of the disease, accompanied the choroidal spots. Another point of special interest in this case is that the amblyopia has been stationary, although the discs have become pale. This case and the last one in the series (No. 15) raise the question as to what is the connection between the changes in the discs and those in the choroid. It is evident that the two do not bear any constant relation to one another in degree, since in many cases where the choroidal disease is most abundant the discs are but doubtfully changed. It may be noted that the arteries supplying the part of the choroid most affected, also supply important twigs for the nutrition of the disc. (Cases 11 to 15.)

7. Some cases of choroidal disorganisation at or near the yellow-spot region in old people, in all probability

caused by hæmorrhage. The history of inflammatory symptoms in several of these cases is interesting, and is perhaps related to the rapid escape of a large quantity of blood into the choroidal tissue. (Cases 16 to 19.)

8. A case in which neuro-retinitis and choroiditis appears to have been caused by erysipelas of the face in an old woman, permanent changes being produced in the discs and yellow-spot region. (Case 20.)

9. A peculiar case of recurrent hæmorrhagic disease of choroid and retina in a young man, affecting only one eye and followed by remarkable recovery of sight. (Case 21.)

Case I.—*Retinitis Pigmentosa—History of diseases of nervous system on the mother's side—No consanguinity—Posterior Polar Cataracts of unusual symmetry and regularity.*

Mary Simcox,* 36, married, is the subject of typical retinitis pigmentosa with posterior polar cataracts, and some myopic astigmatism. The pigmentation is exactly symmetrical, and extends from the equator forwards to the periphery as far as can be seen. In each eye the lowest quadrant is much less affected than the other three, and the inner quadrant the most so. The discs pale and hazy, and the retinal vessels considerably diminished. Large myopic crescent in each. A very beautiful and regular radiating form of posterior cortical cataract (see woodcut). In each eye ten striæ pass outwards from the opaque posterior pole; of these, the largest points vertically down, and the shortest up. They vary in length; the longest of them divide nearly midway between the pole and equator and, again uniting further forwards, inclose a minute oval area of clear lens (or capsule); in one or two, when magnified, a thin central line of white opacity could be seen running from end to end of the oval space so inclosed, and dividing it again into two like the mid-rib of a leaf.

She believes her sight was perfect till after her first confinement 12 years ago. She was very ill for a time, and vision began to fail slowly. Very subject to nervous occipital headaches. For

* In rare diseases of long duration, the subjects of which often go from one hospital to another, it is advisable, in order to avoid the multiplication of cases, that the names of the patients should be given in full in journals the circulation of which is strictly limited to professional readers.

the last three years has had a constant "roaring" in the right ear; never in the left. Not deaf, and has never had otorrhœa. She has seen muscæ and "coloured rings and serpents" for some time.

Posterior polar cataract, with leaf-like radiating opacities (magnified and half-diagrammatic).

Vision $\left\{\begin{matrix}\text{right, 12 J.}\\ \text{left, 18 J.}\end{matrix}\right\}$ slowly. Ps. act well.

Vision worse both at night (retinitis pigmentosa) and by very strong light (cataracts).

Family history.—All the relatives are known, and full inquiries have been made up to her father's paternal grandfather. There is no known consanguinity of marriage in any generation, but on the mother's side we can obtain a history for only one generation.

Mother's side.—She was one of about five; the precise position in the family not known for all of them;—

Sister (patient's mother) married at 30; 2 children.

1 (f.), the patient, *Mary S.*, born soon after marriage; well grown, good features, intelligent. Hair, dark brown.

2 (m.), born 13 years later (husband having been at home all the time); died æt. 3 months.

Sister, married, and had 8 children.

5 are living and well; 1 died in infancy; 1 was killed.

1 (f.), (1st cousin to the patient) is married, and has since become liable to frequent fits.

One of the sisters in this family lost a child æt. $2\frac{3}{4}$ (patient's 1st cousin), from "insanity after a fright."

Brother. Married and had 5 children.

3 are living and well.

1st. (f.), (patient's 1st cousin) became subject to fits after infancy, and died æt. 12 of "inflammation."

5th. (m.), (patient's 1st cousin) the youngest, died insane æt. 25. His sight was good.

Brother. Married; has had 10 children. No facts of interest.

Brother. Married; 3 children, all well.

On the *father's side* the only facts bearing in any degree on the case are the following:—

1. One of the patient's paternal uncles died æt. 45 of "fits following delirium tremens;" the fits attributed to a fall from a horse.

2. The eldest child of another paternal uncle (patient's 1st cousin) had infantile convulsions, "her legs never grew below the knees, and she has never walked."

Case II.—*Retinitis Pigmentosa in a brother and sister—History of insanity and defective sight in the family on mother's side for two generations—Marriage of first cousins, but no evidence that it has caused the disease.*

William Allen, æt. 24, is the subject of typical retinitis pigmentosa, with at present only very scanty pigmentation; the changes in the disc and retinal vessels being more advanced in proportion than would be expected. His choroids are extremely pigmented, and probably this fact accounts for the defect of sight, and especially the night blindness, being somewhat greater than the stage of the disease indicates. He has never been able to see well at night, and is getting slowly worse. No deafness. Refraction normal. Black hair; brown irides; well-grown and of florid complexion; intelligence good. Married 7 months ago.

Sarah, 17, his sister, florid, well-grown, and inclined to be stout, intelligent, but excitable. Did not notice any night blindness till about 6 months ago, when she began dressmaking. Typical retinitis pigmentosa, with pale, slightly hazy discs, and much diminished retinal vessels. No opacities in vitreous, and no cataract. The pigment lies in a zone somewhat behind the equator, the parts of the retina in front of this zone, as well as those between it and the *o.d.* being perfectly free so far as can be ascertained; the zone is narrowest and the pigment least abundant, at the lowest part. No deafness. Refraction My. $\frac{1}{20}$.

The family history is long and complicated, and will be best appreciated by giving the pedigree.

First cousins.

Grandmother—Grandfather; he died insane in old age.
A half-sister of his became blind and died paralysed and insane, æt. about 30.

They had seven children, though not exactly in the following order:—

Son. Married, and has healthy children.
Son. „ „ „ „
Daughter. „ „ „ „
Daughter. „ „ „ „
Daughter. Unmarried; half imbecile.
Daughter (the youngest). Married to a 1st cousin. They have children, some of whom are said to be defective in intellect (evidence doubtful).
Daughter (now *Mrs. Allen*). Very "short-sighted," but can see well; not examined. Not related to husband by blood. Has children as follows:—

1 (f.), æt. 26. Thought to have slight defect in sight (could not be examined).

2 (m.), æt. 24. The patient, *William.* Retinitis pigmentosa.

3 (m.), æt. 22. } Nothing amiss.
4 (f.), æt. 20. }

5 (f.), æt. 17. The patient, *Sarah.* Retinitis pigmentosa.

6 (m.), died æt. 6 years. "Water on the brain;" "had no bones, and could not walk unless splintered up to the waist." Head not very large. Was intelligent. Probably paraplegic.

7 (m.), died æt. 6 months. "Inflammation on the chest."

8 (m.), æt. 12. } Nothing amiss.
9 (m.), æt. 10. }

10 (m.), æt. 8. "Very peculiar and excitable," "brain too active and head rather large." Any little excitement is apt to make him delirious and disturbed at night.

Case III.—*Retinitis Pigmentosa, beginning in boyhood—The same defect in two brothers; the three affected brothers being the only survivors in a family of eighteen children—Mother similarly affected, and subject to fits—No consanguinity known.*

George Pearce, æt. 33, came to the hospital in *May*, 1873. He was a healthy looking man, with brown hair, and abundant

whiskers. He said that he could not remember ever having been able to see well at night, and that his sight had steadily got a little worse year by year; he was still able to act as assistant to a bookseller.

Ophthalmoscopic Examination.—Media clear. Conditions precisely similar in each eye. Optic disc pale, grayish yellow; its surface hazy, and semi-transparent ("waxy"). Retinal vessels very small, and only visible for a short distance beyond the disc. Abundant peripheral pigmentation of each retina; the pigment accumulations were highly characteristic, consisting of stellate, linear, and variously branched figures, mingled with which were some black dots. The pigment formed a sharply defined zone at the periphery, and was not visible unless the patient looked considerably to one or other side of the observer's eye; it was much more abundant at the upper and outer part of the affected zone in each eye than in any other portion. There was no evidence of choroidal disease, excepting slight pallor and irregularity of its pigment close to one part of the edge of each disc, and in one eye two or three small white, ill-defined dots in the choroid near the disc, doubtfully pathological.

Family History.—The patient knew nothing of any relatives further back than his parents; he said he was certain that his father and mother were not blood-relations. His mother had bad sight from his earliest recollection, and eventually she became "quite blind;" she could at first see better by day than at night. She was subject to "epileptic fits" from the age of about 18 upwards. She had 18 children, of whom 15 died, as the patient believed, in infancy. The eldest living was the first born, and the two next (of whom the patient was one) were either the second and third born, or at any rate came quite early in the family; they all had bad sight. All are males.

1 (m.), æt. about 40, said to have been "quite blind" for about eight years, and had bad sight for long before.

2 (m.), æt. about 37; now nearly blind.

3 (m.), æt. 33, *the patient*, reads 16 J. in the daylight, but can see scarcely anything after dusk. He shows no signs of hereditary syphilis; unfortunately I have no note about the acquired disease, though from his having for twelve years had a perforation of the soft palate, it is almost certain that he has had the disease. Has been married ten years, and has several children, in

some of whom there is a history suspicious of inherited syphilis. I have noted no deafness in the patient, and should hardly have omitted it had it been present in any marked degree.

CASE IV.—*Retinitis Pigmentosa—Failure of Sight not beginning till æt.* 35 *—Pigment rather scanty—A half-sister reported to be similarly affected—Probable inheritance from Father in both cases—No consanguinity known.*

Betsy Smith, æt. 52, came under care in *February*, 1874. She dated the failure of her sight from a severe illness, "nervous debility" about 15 years before; she believed it had been perfect till she was about 35. She could still see to read large print, and to write, with her + 20 spectacles. She was of small stature, old looking, and feeble; had lost many of her teeth, and had for years suffered much from indigestion, probably from bad living.

The optic discs were slightly hazy, and of a yellowish white colour; retinal vessels extremely diminished; the retina was pigmented at the periphery, but the changes in this respect were not so far advanced as the state of the retinal vessels led one to expect; the pigment deposits were widely scattered, and comparatively few in number; most of them were small, straight lines of black, a few of which presented a small circular enlargement at one end; there were scarcely any spots or patches of pigment, and very few stellate figures. There was slight nuclear haze of the lenses, and some peripheral striæ, but no posterior polar opacity. I noted that the right disc was paler and less hazy than the left. There were no signs of hereditary syphilis.

Family History.—Her father's mother "went nearly blind" in old age, and the patient's father himself also had bad sight when an old man, but no details of the cause of the failure in these two cases could be ascertained. The father married twice, and had two families. One of the patient's half-sisters (her father's daughter by the second marriage) is stated to have the same failure of sight with night-blindness, as the patient. The father is said not to have been related by blood to either of his wives.

CASE V.—*Conditions simulating Retinitis Pigmentosa, with disease of Vitreous and Myopia—Latterly Nystagmus and increasing Amblyopia—Nervous Symptoms, ? caused by Nystagmus or by Cerebral Disease—Insanity in a Brother—Hereditary Syphilis probable.*

Ellen W——, 47, admitted in *October*, 1876, with nystagmus. Hair nearly black. V. about 12 or 14 J., varying with the light. Constant lateral nystagmus, continuing even when the lids are closed. Slight central haze of each cornea. Pulse active, round, and of normal size. Ophthalmoscopic changes symmetrical, but rather more abundant in the right. Lenses clear. A sharply circumscribed floating body, deeply seated in the vitreous of the right; none in the left. Refraction probably myopic, but the oscillation prevented positive proof. Abundant pigmentation of retina in bone-corpuscle shapes, dots, lines, and larger irregular spots. *o.d.* pale and "waxy." Slight choroidal changes near *o.d.* (myopic), but elsewhere no proof of former choroiditis.

The pigment spots were not distributed quite regularly, nor were they so uniform in size and shape as in typical retinitis pigmentosa.

No proof of hereditary syphilis, but physiognomy rather suspicious, and she is the first born. She probably, from the history, had purulent ophthalmia in infancy. Has never been able to smell since infancy (? syphilitic disease). Other special senses perfect. The second child (m.) died of measles, æt. two years. The third (m.) died, æt. 32; insane from "softening of the brain." There were three others by her mother's first marriage, and four by a second marriage, but all died in infancy.

Patient's mother died, æt. 49, in her third attack of "apoplexy." Had been paralysed on left side in one of the previous attacks. Father, a publican, died, æt. 47; insane; it appears doubtful whether it was delirium tremens or not.

She appears not to have been much inconvenienced by her eyes until a year and a half before I saw her. She had been accustomed to do needle work in the dark waiting-room of one of the underground railway stations, and had formerly been able to do the finest work, though always near-sighted. About *May*, 1875, some friends who had known her a long time, told her of the nystagmus. Since that time her sight has continued to get

worse. She has become nervous and restless, and has slept badly; has had much headache, especially in right forehead; and is liable to "confusion of the head," and "so hysterical." The headache has become worse lately, and makes her feel bewildered When walking in the street she is sometimes obliged to stand still for fear of falling; she does not lose consciousness, but feels as if she could not move. It is doubtful whether these attacks are not due to the nystagmus and defective sight. No tremors or weakness of limbs, and no defect of speech. Memory has not failed. Many years ago, "dropsy" all over her; since then, with the exception of piles and habitual constipation, health tolerable. Still menstruates, but irregularly. Married, and has one son, æt. 20, reported healthy; husband died a year after marriage.

Case VI.—*Unsymmetrical Choroiditis—History of Diseases of Nervous System in a Brother and Sister of Patient—Evidence as to Hereditary Syphilis negative but incomplete.*

Rebecca H——, 36, married seven months, and pregnant three months. Had noticed defect of her left eye for only a few weeks before admission in *August*, 1876. There was no evidence of syphilis, either acquired or inherited, excepting in the eye.

V. of *left* = 50 Sn. at 20 feet, and not improved by glasses. Slight general haze of fundus (probably retinal); vitreous appears clear. A very large patch of choroidal disease, rather below and to inner side of *o.d.*; its shape is an irregular oblong, and it is composed of coal-black spots interspersed with some white at the centre (her hair is black and irides brown). It has every appearance of having been in its present state for years.

Right healthy.

She had for years been subject to bad headaches at the top of the head, but they had ceased since marriage. Never any fits. Many years ago was severely poisoned by some arsenical wall-paper, and not expected to live. Two others, young children, not related to her, but in the same house, were affected.

Father died rather young. Mother lived to 71. There were 8 pregnancies.

1 (m.), died, æt. 18. Was an idiot, and subject to fits all his life. Not paralysed.

2 (f.), æt. 43.

3 }
4 } died young, of small-pox.

5 (f.), æt. 40. Was for eight months in Hanwell Asylum, after her second confinement, æt. 33. Has since had several children without a relapse.

6 (f.), æt. 38. Married, and has children. Has been subject to fits since æt. 12.

7. *The patient.*

8. A still-birth, some years after the patient.

CASE VII.—*Amaurosis with Atrophy of Discs and Diminution of Vessels, followed by general Paralysis of the Insane.*

Henry W——, æt. 35, married, a smith. Admitted with the barest p. l. on *April* 26, 1876; can just tell where the window is with each eye. Ps. slightly dilated, but do not expand in the least when shaded; the right is slightly larger than the left. *Left* eye deviates definitely downwards, and a little outwards, but this varies, and sometimes it is simply divergent. Associated movements of eyes appear perfect.

Ophthalmoscopic Examination.—Conditions symmetrical. Gray-white atrophy of discs, with diminution of the retinal vessels. The discs uniformly pale all over, quite clean, and the stippling of *lam. crib.* well seen; slight but definite superficial disturbance of choroid at border of each, especially on yellow-spot side. A small white projection from the inner border of right *o.d.*, possibly remains of neuritic effusion, but more probably congenital. Diameter of retinal arteries diminished by about one-third or a half; veins less diminished. No changes elsewhere.

Detailed examination elicited no other symptoms of ill health, or of nervous disease, excepting that he had slight difficulty in standing when not holding some support. Did not consider his legs weak, but felt rather "rocky" or unsteady. Eats and sleeps well; not too sleepy. Manner reserved and quiet, but intelligent. No constipation.

Sight began to fail six months before admission. The failure had been gradual, and, as he believed, quite symmetrical; in about three weeks from its onset he had become unable to read the newspaper.

For the last six years has drunk a very little beer; for sixteen years previously had been a total abstainer. Until a year ago

(about six months before amaurosis began) had smoked half an ounce a day; since then has diminished it because his work does not admit of it; now smoking only an ounce a week. Once slight gonorrhœa, but no evidence of syphilis. Denies sexual excesses at any time, and does not admit any diminution of power. No history of sun-stroke, or of injury to head or of otorrhœa. No family history of nervous or mental diseases, nor of amaurosis, admitted by him or his mother. Has lost three brothers or sisters from various complaints; others are living. He told me he was unmarried, but I learnt from his mother, after his death, that he had been married since about æt. 20, and that his wife had gone away with another man two years ago. He had no children. It is perhaps, not improbable that, notwithstanding his denial, he had become impotent some time before his sight failed. His mother believed that his wife's desertion was the cause of much inward grief, though he showed no emotion.

He attended for nearly eight months. No pronounced symptoms appeared until *October*, when he became shivery and ill, and had pain in the abdomen for a few days. On *December* 19, there was "decided incoordination in walking, and considerable difficulty in standing with the feet together. He sways about, and has to balance himself." Movements of eyes still perfect, excepting the slight divergence of left. His manner and expression had become rather vacant, but I attributed this to his blindness.

In *January* last, he became subject to repeated "shivering fits," and would creep near the fire. His legs, too, "would sometimes fail under him, and yet he was not paralysed." In *February* he became insane (melancholy), and was sent to the Brookwood Asylum, where he died under the care of Dr. Brushfield on *March* 5, from general paralysis of the insane. Dr. Brushfield has kindly given me the results of the *post mortem;* the membranes were thickened, the arachnoid being quite opaque. Considerable subarachnoid effusion and the ventricles dilated with fluid. The brain presented a peculiar pale and sodden appearance and its substance throughout, but especially the cerebellum, was softened and disorganised. The optic nerves were atrophied. No trace of tumour.

There had, throughout his attendance at the hospital, been a

complete absence of ataxic pains, of pins and needles and numbness, and of subjective tightness either around the abdomen or across the nose. The last time I saw the man (in December), I thought from his gait that he was becoming ataxic. He had no delusions or exalted ideas while under my care.

There was not during his attendance at the hospital any marked alteration in speech or tremor of the lips and tongue. He sometimes answered slowly, and his countenance had a heavy vacant aspect, partly from imperfect closure of the lips; but he answered all questions clearly and accurately. Not improbably a specially skilled observer would, however, have detected some defect in these particulars.

CASE VIII.—*A case of slight and variable Amblyopia with occasional Diplopia—Tremors, Vertigo, thickness of speech and weakness of legs—Intolerance of tobacco and alcohol—Slow advance of the symptoms—Diagnosis; Cerebro-spinal disseminated Sclerosis.*

John C., æt. 47 on admission, a stonemason, has been under care at intervals since *October*, 1874, for varying defects of sight, accompanied by other nervous symptoms. The prominent facts in regard to all his symptoms have been rapid onset, and more or less complete recovery, followed by relapses. The chronological order of events has been about as follows:—

At the end of September, 1874, four or five weeks before I first saw him, while reading the paper one evening, he suddenly complained of headache. Went to bed, and next morning "could not see to judge a stone," and "could not see across the road," having been able to see perfectly the day before. Improved in two days enough to allow of resuming work, but did not recover perfectly.

On admission, October 10th. Pupils small, and "eyes very restless;" reads 4 J. with each. No disease of discs or retinæ visible. In left eye two patches of old choroidal disease, with rounded sharp outlines and much black pigment on a greyish ground, between disc and yellow spot, but above; one as large as *o.d.*, the other much smaller.

Manner peculiar; he seems half-asleep and muddled as if half-drunk; yet is nervous and replies in an irritable, querulous manner. Has always been nervous, but this and the irritability

have increased of late. Appetite good. Has slept well till within the last week or two. Tongue whitened and fissured. Married, but wife never pregnant. Had gonorrhœa 25 years ago; no evidences of syphilis excepting old choroidal disease in the left eye and the condition of the tongue. Some years ago drunk beer and spirits heavily. Has smoked largely (half-an-ounce of shag a-day) for 25 years. A small puny-looking man, of spare habit, with black hair and rather sea-weedy complexion.

Ordered 5 grain doses of iodide of potassium three times daily, and told to leave off smoking, though I did not look upon the case as an ordinary one of tobacco amblyopia.

The urine, tested twice in November, contained neither albumen nor sugar.

November 18.—Worse again since yesterday. V. to-day = 6 J. 28*th.*—V. = 4 J. *December* 5*th.*—V. = 2 J. easily. 19*th.* The same.

He then went to work in the country till end of *April*, 1875, when he returned to London.

On *May* 1*st* he came to be seen. "V. = 1 J., with some difficulty. Health good." Remained perfectly well till about middle of July, when his wife died unexpectedly, and he was much upset.

August 7*th.*—V. = only 6 J. with each separately. Attributes this relapse of amblyopia to shock above-mentioned. Since the beginning of the year has again been smoking a little (about half-an-ounce a week), but V. remained unaffected till the above event. *Ordered* bitter mixture.

14*th.*—Complains of frequent variations of sight; it sometimes varies every two or three hours while he is at the same piece of work; it is always worst in the evening. It seems to depend on failure of accommodation. V. to-day each eye = 1 J. slowly, p. 12″, and $\frac{20}{30}$; not H. m. $\frac{1}{50}$. Ordered + 36 glasses.

Now ceased attending for more than a year.

October 17*th*, 1876.—Sight again failing for the last fortnight. Has never quite left off smoking since he resumed it in January, 1875. No trouble or worry lately. Manner as at first.

November 4*th.*—Still complains of variations in the sight.

11*th.*—V $\begin{cases} \text{Right, 6 J. at 12″ and } \frac{20}{40} \text{ or } \frac{20}{50} \\ \text{Left, 10 J. and } \frac{20}{50} \text{ badly.} \end{cases}$

Ps. active. Complains of occasionally seeing double, but cannot give an accurate account of it.

Ophthalmoscope.—Right o.d. probably healthy, perhaps slightly gray on yellow-spot side. *Left*, decided pallor and atrophy, on yellow-spot side. Is at work, and earning full wages. Iodide and nux vomica in small doses.

18*th.*—"On 16th had to leave off his work on account of bad sight. Yesterday it was well again, and to-day remains well. The work was exactly the same as usual. Says he had double sight for distant objects. Movements of eyes now perfect in all directions, excepting that there is slight defect of convergence."

December 16*th.*—Has had no more attacks.

May, 1877.—Remains pretty well in his own opinion; but is liable to frequent attacks of giddiness. Has not lately had any attacks of diplopia or dulness of sight. He is more tremulous and complains, when asked, of considerable weakness of legs, shown by soon tiring at work or when walking. Manner much as before. I may note here that I was often in doubt whether his quasi-drunken manner was partly the result of alcohol or wholly due to his disease. Sometimes he admitted that he had had some drink, but added that he was now affected by such a small quantity that he scarcely knew how to drink little enough; the last time I saw him he said that a glass or two of beer would now make him nearly drunk, and that he had almost given up taking it.

Case IX.—*Localised Exudative Choroiditis with Retinitis near Y.S. in one Eye only, and accompanied by Keratitis Punctata—Recovery of nearly perfect V., with partial Atrophy of Choroid—Patient a delicate lad, æt.* 17; *Family Gouty and probably Tuberculous—Possible influence of Toothache.*

Thomas H., æt. 17, a shoemaker's apprentice, came on *December* 11, 1876. His right eye had, as he believed, begun to fail two or three days before, without pain or other symptoms. There had been some toothache in the carious right upper central incisor for a few days before the dimness was noticed.

V. { Right, 8 J., barely, not nearer than 12—14″, and $\frac{20}{200}$.
{ Left, 1 J. well, p. 4, r. 12″, and $\frac{20}{20}$.

Ps. active, but right remains always larger than left. Movements of eyes perfect.

Right Eye.—Numerous very minute dots on posterior surface of cornea, in its lowest half only; no haze; no congestion; no iritis. *Vitreous* clear. *O.d.* very pale and hazy, the haze extending for some distance into the retina, especially inwards and outwards; some of the smaller vessels partly veiled but the larger ones all clear. Retinal veins too large, especially the (real) lower one; arteries, n. Close to the *y.s.*, rather below and to its (real) nasal side, a large patch of bluish-white effusion, most dense at the centre and thinning off at the margin; it is crossed near its centre by two small retinal vessels which are quite unobscured, showing that the overlying retina is clear. No pigment disturbance around this patch. Refraction on the patch $= H_{\frac{1}{24}}$; at another part of retina, equally distant from *o.d.* $= H_{\frac{1}{48}}$ at most; thus the patch is slightly swollen. *O.d.* not much swollen. No hæmorrhages, nor any patches of effusion elsewhere. The patch might be taken for a choroidal tubercle, but is bluer or less yellowish than the recent tubercles I have seen during life.

Left eye, fundus n.

Urine.—No albumen and no sugar.

He took iodide of potassium in 5 grain doses three times a-day, and a mercurial pill once a-day, containing 2 grains of gray powder, until the end of *January*, 1877 (eight weeks), without salivation or other disturbance.

January 20th, 1877.—*Right* eye, V. = 2 J. at 6″ slowly. The patch now sharply circumscribed, and the haze of retina and *o.d.* has disappeared.

April 25th.—V. right, 1 J. r. = 14 inches, as left 1 J. easily, as with left well.

Occasionally notices a little spot in the middle of the right visual field, but on trial no scotoma can be found to-day. The seat of the former effusion is now represented by an area of partially atrophic choroid considerably larger than the *o.d.* Over the greater part the atrophy is incomplete, and the appearance seems caused by denudation of the pigment epithelium, with here and there little pigment heaps; the centre is whiter, and indicates more complete atrophy. *O.d.*, slightly pale; retinal vessels n.

If the pigment epithelium is removed or seriously disturbed

over the whole patch it is difficult to understand the absence of a large nearly central defect of sight. It may be suggested that the appearances are due to various degrees of atrophy of the choroidal stroma, such as would probably occur after caseation and absorption of a tubercle in the choroid if the patient were to live long enough. In such an event the choroi-capillaris and retinal epithelium might escape with little or no damage.

The personal and family history gave no definite clue to the cause or nature of the disease. There was no reason whatever to suspect acquired syphilis. No evidence of hereditary syphilis in the patient, but the family history suspicious. Patient was very delicate and undersized until about three years ago, when he began to grow fast; he is now slightly built, of moderate height, with fair hair, and he looks delicate. Is not known to have had any illnesses, is not subject to epistaxis. Believes he had a slight blow on the right eye about six months before it failed. Masturbation denied. Mother married at 18, had 9 or 10 children, and died at 40 of "consumption." Father married at 21, and died soon after 40 from "liver disease caused by drink;" he is known to have had several attacks of gout in his toes. Of the 10 children 3 are living; one (f. æt. 32), another (f. æt. 28), and the third the patient. The two or three eldest and several born subsequently died at from one to three years old of "water on the brain." The patient is about the youngest. Thus the history, though suspicious of syphilis, points quite as strongly to tubercular disease and gout.

Case X.—*Localised exudative Choroiditis at Fundus of one eye, implicating overlying Retina, and leaving Absorption Patches comparable in appearance to Herpes Scars—Recovery of nearly perfect V.—Relation of the Choroiditis to Retinal Vessel—Patient, a girl æt.* 14, *Strumous—Possible influence of over-use of eyes.*

Alice W., æt. 14, admitted in *October*, 1876, with dimness of the left for the past two weeks, and asthenopia of both from too much needlework. Slight H. was found, and she was ordered + 40 spectacles with benefit.

In the left eye, at some distance from the *o.d.*, below and

rather to its nasal side (erect) was an extensive, recent, exudation patch obscuring a large descending retinal vein; the disease had the appearance of localised retinitis, the underlying choroid being hidden.

Several months later I saw her again. She was getting on fairly well at millinery work with her + 40. V. right 1 J.; left 1 J. barely.

The character of the changes in the left fundus had now completely altered. All haze of the retina had cleared off, but the corresponding part of the choroid had undergone pronounced atrophic changes extending through its entire thickness; the retinal vein, which had at first been obscured, running across the middle of the diseased area. The choroidal atrophy was arranged in two rounded patches joined by an isthmus, and so having a sort of figure of 8 shape, the head of the figure being the smaller and nearer to the *o.d.*, the lower patch being about as large as the *o.d.* itself. On closer inspection the diseased area was seen to be composed of numerous much smaller round spots, which, having become more or less confluent, had together given rise to the above figure. Each spot was about twice as wide as the overlying retinal vein. There were no separate or outlying spots, all were clustered close together. The individual spots could be distinguished as being separated from each other by collars of pigment, more or less distinct, and the whole appearance reminded one rather forcibly of a patch of minute herpes vesicles. Here and there a large choroidal vessel could be seen crossing some of the spots, but the white sclerotic was well exposed over the greater part of the diseased surface. No changes elsewhere.

She is strumous, with thick nose and very sluggish circulation; liable to soreness inside the nostrils, and suffers very much from coldness of hands and feet; cheeks very florid and patchy. Not liable to herpes labialis. She is the third in a family of six; no signs of hereditary syphilis. A short time before the eye failed she had been apprenticed and begun to work hard at millinery; this had brought on the accommodative asthenopia noted above, but there had been no other symptoms, nor had the left ached more than its fellow. She had not caught cold or been out of health when the eye failed. Both parents living and reported well, but residing at a distance and not seen.

CASE XI.—*Central guttate Choroiditis followed by well-marked changes in Optic Discs—Case under notice* 16 *months—The Amblyopia preceding the changes in the Discs and not increasing when they appeared—Patient senile, and for years a considerable beer-drinker—Attack of Apoplexy followed by Mental Derangement.*

James H., æt. 58, a traveller, admitted in *January*, 1876. A man of large frame and of good muscular power, but very gray, and aware that his memory was failing; acne rosacea of nose and cheeks. Sight had been failing for three months, and he dated it from his wife's death.

V. with each eye = 16 J., and with + 10 or + 8 = 10 J. slowly; cannot see 200 Snellen at 20 feet. *Ophthalmoscopic changes*;—The conditions are symmetrical, with slight differences, and the changes confined to the region of the *y.s.* In the *right* are some small, dull whitish or yellowish dots scattered over the region of the *y. s.*, but more at its circumference than close to the *fovea centralis*; easily overlooked by indirect examination. In the erect image they are approximately circular, and surrounded by a decided, though slight, collar (often incomplete) of pigment. In the *left* are two or three little groups of spots like the preceding, but placed between *o.d.* and *y.s.*, the latter part itself being free from them. In this eye the groups are composed of spots which are more or less confluent; they are whiter than those in the right, and perhaps rather more pigmented; there are, however, a few quite isolated, one being bright white and another a minute black dot. *Discs* perhaps very slightly hazy; no pallor and no cupping; retinal vessels normal. *Urine* pale, nearly clear; no albumen.

April, 1877; he considers V. just the same; with his + glasses *right* = 10 J. barely; *left* = 10 J. tolerably well. *Ophthalmoscopic appearances*:—The spots in the right appear to have increased in number, there being now several little groups, each composed of two or more single dots which have become confluent, and at one part there is denudation of the pigment epithelium over a rather large area containing several of these groups. In *left* they are not quite so abundant. In neither eye are there many singly isolated dots, nor do they extend to the nasal side of the disc. *Discs* both pale; the *right* is quite decidedly pale all over, but more so towards the *y.s.* and the sclerotic sheath easily seen;

in the *left* the pallor is less marked on the *y.s.* side and scarcely present at all on the nasal side; retinal vessels normal. Thus the choroido-retinal disease appears to have increased somewhat at least in the right eye, while the discs have certainly undergone marked change, without any appreciable increase of the visual defect. On both occasions the pupils were dilated with atropine. Refraction hypermetropic.

He had through life had good health. Nothing in the family history threw light on the case. No reason to suspect syphilis either remote or recent. Some years ago had an attack of inflammation of both feet, which he took for gout, but has had no recurrences. Fond of beer, and in his trade obliged to take it freely, but has seldom if ever been drunk; has not drunk spirits. Is a smoker. In *March*, 1876, soon after admission, he had an apoplectic fit while at stool; was unconscious for about a day, and "out of his mind" for several weeks. After this he had "rheumatism" in the left shoulder, much pain in the bone; it did not affect any other parts. Had no paralysis after the fit, and considers his recovery to be perfect, with the exception of an increased weakness of memory. He is, however, now (*April*, 1877) decidedly feebler and older looking than before the attack. His radial arteries are not tortuous or hard.

Case XII.—*Guttate Choroidal Disease in the Y.S. region in an elderly woman—Nuclear and Posterior Polar Cataract of this Eye and nearly complete Cataract of other.*

Isabel G., æt. 59, admitted in *November*, 1876. *Right* cataract nearly complete; V. = fingers at 12 inches. *Left* V. = 14 J. barely at 12 inches; + 10 = 6 J. fairly well; after dilatation of pupil by atropine,—some nuclear haze, and considerable patchy opacity at posterior pole of lens; some little dots of whitish choroidal disease between *o.d.* and *y.s.* Complete examination impossible, from the state of the lens. Urine free from albumen. Is still under care, and the amblyopia has advanced but very little.

Case XIII.—*Choroidal Disease with pale Discs in an old Woman—Peculiar dotted Cortical Opacities in the Lenses.*

Elizabeth C., æt. 77. Her sight had been for about a year as defective as on admission. She had in her time done a great deal

of fine work. V. right 15 J.; left 20 J., and 70 Sn. at 20 feet. Pupils fairly active. After atropine, focal light revealed a number of small round dots of opacity in the cortex of each lens; they were not white, but of a duller tint. *Ophthalmoscopic examination* showed an area of choroidal disease in each *y.s.* region, larger and and more marked in degree in the left than the right. The morbid changes had produced an ill-defined mottling or coarse pepper-and-salt appearance, which, on closer inspection, was resolved into little whitish patches or spots separated by small irregular accumulations of pigment. There were no peripheral changes. Optic discs slightly pale.

Case XIV.—*Superficial changes in Choroid, or in the Pigment Epithelium, near O.D. and Y. S. in an old woman—Posterior Polar Cataract of same Eye, and complete Cataract of other.*

Sarah B., æt. 65, came under notice in *August*, 1874. The condition of her eyes when admitted was as follows:—*Left* V. = shadows; mature white cataract, believed to have been coming on for a year. *Right* V. failing about two months; with her + 11 glasses = 10 J.; *after atropine*;—slight general haze and decided opacity of posterior surface of lens, especially at the pole; *o.d.* surrounded by a dusky ring; choroid remarkably patchy, large areas of it being of considerably lighter colour than others, but no spots of pigmentation or absorption. The *y. s.* region was obscured by the polar opacity of the lens. The evidence of choroidal disease here was but slight, and it may have consisted merely in senile atrophy of the pigment epithelium.

She remained under care for more than a year, the cataract in the right advancing slowly. When last seen she was barely able to work, but was averse to operation.

Case XV.—*Amblyopia coming on in association with Paraplegia many years ago—Recovery from the Paralysis—Amblyopia stationary, with incomplete atrophy of Discs, and a spot of central Choroiditis in one Eye—Patient smoking throughout—Symptoms attributed to nervous shock.*

Edward B., æt. 55, formerly a soldier, now a traveller in the paper trade, came for advice *March* 15*th*, 1876. Intelligent, nervous, and fussy (a bad witness), very gray, and looking several

years older than he is, but still strong and active. A smoker, but does not drink enough to cause symptoms. No history of syphilis, but once had gonorrhœa. His father and a brother reported to have had gout; he himself has had neither gout nor rheumatism.

The history of his amblyopia was difficult to get accurately, but appears to have been substantially as follows:—He served in the Crimean war, and had slight "fever and ague," but his health was not broken by it. Remaining in Constantinople after the war, the house in which he lived was burnt down in the great fire, and he was much frightened. After this, and as he says, in consequence of the fright his legs were paralysed and he was obliged for a time to go about on crutches, but in the end recovered perfectly; there was no numbness nor any affection of the sphincters. During the paralysis his sight failed. His account is not definite, but it appears that both eyes failed together, that the amblyopia reached its height in a few days or weeks, and that it had ever since remained stationary. He came to the hospital because advised, and not from any recent failure of sight.

On admission V. (without glasses) each separately = 16 J. slowly at 6 inches, and 200 Sn. at 20 ft. H.m. $\frac{1}{30}$ = 100 Sn. at 20 ft. With +11 reads 8 J. slowly at 6 inches. Ps. do not dilate fully with atropine, and the left becomes oval upwards.

Ophthalmoscopic Examination.—Media clear. Discs pale all over, but much more so on *y.s.* side where the stippling of the *lam. crib.* is very evident. Retinal vessels normal. In *right* eye, between *o.d.* and *y.s.* a single, small, oval spot of whitish choroidal disease exactly resembling the spots in Case XI above described; it is slightly pigmented on one side (? shadow thrown by an elevation). No other spots in this eye, and none in the other; no disease in any other part of the fundus. Repeated and very careful examinations confirmed the above description.

May, 1877. V. exactly the same. Is better in health and less nervous, probably from improved circumstances. Smokes a quarter of an ounce a day. Hands still somewhat tremulous. Drinks only an occasional glass of beer.

The questions in connection with this case are—

1. What was the cause of the paralysis, and the relation between it and the amblyopia?

2. In what relation does the spot of choroidal disease in the right eye stand to the atrophy of the discs?

In regard to the first, while noting his own impression as to the effect of the fright, it is to be observed that he was an unmarried discharged soldier of nervous temperament, and (judging from his manner) probably of licentious habits. It is not unlikely, therefore, that the sexual system may have had a share in the production of his symptoms. There was no history of sun-stroke. The influence of tobacco in this case is at least very doubtful, although I believe there are some who suspect that this poison may have a definitely injurious influence on certain other parts of the nervous system than those connected with the optic discs.

It is impossible to reply to the second question on the basis afforded by this case alone. Supposing it granted that the presence of the single spot of choroidal disease in one eye is not a mere coincidence, we have the evidence afforded by the preceding case (No. XI) that partial atrophy of the discs may follow the guttate disease of the choroid. The want of a constant relation between the two events is puzzling. Do the punctate changes in the choroid at the fundus stand in any connection with disease of the short ciliary arteries which enter the eye in the neighbourhood of the disc? and may certain senile changes in these arteries be associated with similar processes in the branches which nourish the optic discs?

Case XVI.—*Extensive Choroidal disease with opacities in Vitreous in one Eye—No cause ascertained—Failure described as recent and accompanied by much pain—Other Eye Leucomatous.*

Anne M., æt. 60, came to the hospital on *April 7th*, 1874, with the complaint that her right eye had been failing for 5 months, previous to which time she could see to sew with it. She said that she had had great pain in it when the failure began. The pupil was motionless; T. n.; cornea clear; V. barely shadows. The other eye was much damaged by a large old leucoma, the result of ulceration.

On examining the right, after atropine, I found many large floating opacities in the vitreous, together with numerous very large patches of atrophied choroid, on which there were slight accumulations of pigment; the eye oscillated, and the details of the fundus could not be satisfactorily seen, nor did I ascertain the refraction. It seemed probable that the recent failure was due chiefly to the opacities in the vitreous. I did not succeed in getting any evidence as to the cause of the choroidal disease.

CASE XVII.—*Extensive disorganisation of Choroid in Yellow-spot region with opacities in Vitreous of same Eye—Sudden onset—Probably Choroidal Hæmorrhage.*

Jacob W., æt. 43, a gardener, stated that in *July*, 1872, his *left* eye became "blood-shot," slightly painful, and very dim; he considered that the sight continued to get worse for about 6 weeks. He then had advice at an ophthalmic hospital, and the sight began to improve. Finding the distance too great, he came to the South London on *January 11th*, 1873, when it was found that the left vitreous was so cloudy that the fundus could not be seen. For the next 2 months he took iodide of potassium with citrate of iron. On *March 15th* his vision with right = 1 J., with left = 6 J. Ophthalmoscopic examination of the *left* showed many very thin floating films and minute dots in the vitreous; and at the fundus, rather above the yellow spot, a large area of atrophied choroid, the stroma being atrophied, but not quite equally in all parts; the entire patch was of irregular shape, its boundary everywhere sharply defined, and in many parts forming portions of a circle. On one part of the atrophic area were two or three coal-black blotches of considerable size and irregularly stellate shapes; the remaining, and much the larger, part of the diseased area was free from pigment, excepting a slight sprinkling or dusting of minute dots here and there. There were no signs of past or present iritis. The vitreous still remained cloudy 3 months later (*June*), since which he has not attended.

With reference to cause in this case we can only speculate. My notes are unfortunately deficient in containing no reference to syphilis, but the history and appearances are very unlike disease of choroid or retina due to that cause. His account was

that the dimness of sight began on the evening of a hot day when he had been working with a threshing machine; he thought at first that something had blown into the eye. When at its worst the sight of this eye was so bad that he could not see people. A large hæmorrhage into the choroid breaking through into the vitreous is the most likely explanation of all the symptoms and changes.

Case XVIII.—*Disease of Vitreous and of Retina and Choroid near Y.S. of one Eye only—Probably indicative of previous Hæmorrhage.*

Daniel C., æt. 67, admitted *July*, 1875. V. *right* = 16 J.; + 40 = 100 Sn. at 20 ft.; *left* not 20 J. *Right*, commencing nuclear haze of lens and some opacity in the anterior layers; fundus obscured, but, so far as it can be seen, appears healthy. *Left*, no opacity of lens; a few small, sharply defined opacities in the vitreous; some minute white dots (probably retinal) in the *y.s.* region, and above the *o.d.* (in real position) a large area of indefinite pigmentation situated beneath the retinal vessels.

The appearances here were such as might have been left after a large hæmorrhage, and the want of symmetry is another point in which the disease in this case differed from the previous series (Nos. XI to XV). He only came once.

Case XIX.—*Permanent and very pronounced Retinal Changes in one Eye of an old woman; probably following extensive Hæmorrhage at the Y.S.*

Hannah P., æt. 77, has central whitish-brown opacity of her *right* cornea of many years' duration, and preventing a view of the fundus. About 3 years before admission the sight of the other (*left*) eye failed rather quickly. She gave the history of some inflammatory symptoms. After the use of atropine I found in this eye some striæ in the anterior layers of the lens. The *y.s.* itself was occupied by several rounded, reddish-black spots of large size; these were bounded towards the *o.d.*, and above and below, by a wide crescentic tract of bright white deposit, which could be seen at its margins to be made up of innumerable, small, round dots, while its central parts seemed almost homogeneous. This horse-shoe of white was open towards the temporal

side. The *o.d.* itself was unaltered. She had for many years had a bronchocele. I saw her only once; but a year later heard that she was in good health, and her sight about the same.

CASE XX.—*Changes left by past Neuro-retinitis, probably caused by Erysipelas invading the Orbits—Patient an old woman.*

Susan C., had severe erysipelas of the face, the eyelids being closed by the swelling, when æt. 62. Up to the date of the attack she could see to thread a needle, but ever since recovering from it her sight has been so defective that she has done scarcely any needlework. It remains uncertain whether the amblyopia reached its maximum immediately, or whether it has continued to progress ever since.

August, 1875. It is 3 years since the erysipelas. Her memory is bad, and it is uncertain whether there has been any recent failure. V. = some letters of 20 J. with both eyes open; rather better with left. Pupils rather large for her age, and act moderately; they dilate fully with atropine. Media clear. Ophthalmoscopic appearances alike in the two eyes, excepting that the pallor of disc and contraction of retinal vessels was more marked in the right; *o.d.* pale and rather hazy; retinal arteries and veins considerably diminished; *y.s.* occupied by a patch (about one-quarter the area of the *o.d.*) of dark purplish-black pigment, abruptly defined and with little marginal projections, like the rifts which are so often seen radiating from the *macula* in prepared specimens of this region; choroid between *o.d.* and *y.s.* thinned, patchy, and whitish; a few small pigment dots on nasal side of *o.d.* No changes elsewhere.

At an examination six months later the conditions were the same, but I noted that the central veins on the disc were smaller than the arteries. There was no cupping.

The urine contained no albumen. She had had neither fits nor paralysis, but was in a very feeble and senile condition.

Case XXI.—*Relapsing Hæmorrhagic Neuro-retinitis and Choroiditis, with disease of Vitreous and Keratitis punctata—Only one Eye affected—Remarkable recovery of Sight after repeated attacks—Liability to dimness of Sight of same Eye with Dyspepsia, &c. (? functional)—? Influence of Neuralgia of 5th in determining one of the Hæmorrhagic attacks—Rheumatism and perhaps Gout in family.*

Alfred U., æt. 32, a warehouseman in the leather trade, admitted *December 3rd*, 1873. Black, rather curly hair, thin, pointed features; tendency to acne with congestion, on the face; married, and the father of five children. About 5 weeks before admission a cloud came over the left eye, reaching its maximum in about a week.

On admission. Left eye. Visual field very defective; sees 12 J. barely and 70 Sn. at 20 ft. *After atropine*,—media clear; abundant and widely spread choroido-retinitis. The disease appeared to be of various dates, but for the most part recent. The choroid showed numerous patches, irregular in size and shape, of yellowish, greyish, or dusky colour, with milky haze of the overlying retina; a large irregular patch of greyish or dusky colour occupied the *y.s.* region; *o.d.* hazy, especially at its (real) lower and outer parts. At the periphery of the fundus some small patches of absorbed choroid of old date. The great defect of sight was due to the serious changes at the *y.s.*, much of the more peripheral disease being apparently of much earlier date. I did not at this time suspect the hæmorrhagic nature of the disease, and was not sanguine about the man's prospect. Refraction was tested later, after full use of atropine, and found to be H $\frac{1}{60}$.

Right eye: old corneal nebulæ partly marginal, partly nearly central. V. 4 J. and 50 Sn. at 20 ft. Refraction my. about $\frac{1}{16}$. Careful ophthalmic examinations at various dates never showed a trace of disease in the fundus or vitreous of this eye, nor was it throughout, so far as he knew, ever subject to the attacks of temporary dimness so common in his left. On one occasion, however, the retinal veins at the upper and lower parts of the periphery were very tortuous, and one of them considerably larger at the periphery than near the *o.d.* This enlargement subsided progressively, though rather quickly, at some distance from the *o.d.*

Personal and Family History.—There was no reason to think that he had acquired syphilis in any manner, nor was there anything in favour of syphilitic inheritance. He was of nervous temperament, but appeared steady, and denied having ever given way to sexual abuses of any kind. Never liable to epistaxis. He easily flushed up if nervous or excited. In boyhood he had a severe inflammation of both eyes, lasting more or less for three years, no doubt accompanied by ulceration, and giving rise to the permanent nebulæ on the right cornea. His father had several attacks of "apoplexy," ending in hemiplegia, and finally died of a seizure, æt. 59. Two of patient's brothers have had rheumatic fever severely; one of them (the eldest of the family) is said to have been paralysed on one side during the fever, and now has some defect of his right eye. The only suspicion of gout lies in the history that the father (who was in the leather trade) once had to cut his boot for inflammation of one great toe.

Ordered iodide of potassium and bichloride of mercury, in doses of 5 grains and $\frac{1}{16}$ grain respectively; this made him sick, and in a few days some cinchona was added. On *December* 10*th* (one week after admission) he thought his sight "much improved." *January* 10*th*, 1874, he complained so much of sickness from the medicine, that half doses were ordered, and on 28*th*, the sickness being no less troublesome, he was told to omit all medicine for a time. The iodide had brought out copious acne on the face. On *February* 14*th* vision of affected eye = $\frac{20}{40}$, and 1 J. at. 8″. The optic disc at this date was clear, perhaps slightly pale, and the retinal arteries I thought slightly diminished; subsequent examination made this doubtful.

I saw nothing more of him until *April*, 1875, when he came at request. At this date (more than two years since last attendance), he could still see 1 J. well with the left eye. *Ophthalmoscopic examination* showed a number of atrophic and pigmented patches of old choroidal disease in all parts of the fundus; several of them were close to the yellow spot. The atrophic patches seemed not to be so numerous as the patches of recent disease had been on his first admission, which would appear to show that some had been absorbed without causing disorganization of choroid. The optic disc and retinal vessels were healthy.

September 29*th*, 1875.—Applies again, complaining that during the last three months the sight of the left has now and then

become dim for about an hour; near objects look as if in a mist, distant ones remain clear. After the dimness has begun the upper lid droops, and the eye is half shut. Then all the symptoms pass off. It is "like a rush of blood to the eyes." It sometimes comes on after stooping, but sometimes without. The dimness never affects the other (right) eye in which there is the slight, old corneal opacity. Has for long been liable to flatulence; it was quite well while he was under treatment by iodide and mercury; it is a family complaint. Formerly liable to bilious attacks, with vomiting, but of late years nearly free from them; they were not accompanied by headache. The attacks of dimness are not accompanied by either redness or pain in the eye.

At this date V. was good. Right (corneal nebula) = 4 J. Left = 1 J., r. 9″.

He took 2 grain doses of iodide with ammonia and cinchona for two and a-half months (to *December* 11*th*), and double that dose for two months more (to *February* 9*th*, 1876). During this time his acne improved gradually, and he had no flatulence and no attacks of dimness. About the middle of *February*, 1876, he, however, again became constipated and flatulent, and within a few days had three attacks of dimness. The first of these came on quite suddenly, as he was changing his coat; everything was in a fog, and he "could not tell a man from a woman." The next two were much more brief in duration. He was at this time still regularly taking the iodide in 4 gr. doses.

On *January* 8*th* I noted that there was decided though slight haze of *o.d.* and surrounding retina, with fulness of retinal veins. The changes in the choroid are for the most part superficial. *February* 19*th*.—Left eye well again, and reads 1 J. fairly at 12″.

February 19*th*.—The dose of iodide increased to 7 grains, three times a day, and continued till *May* 13*th*.

March 11*th*.—Has had severe neuralgia in various parts of the head. No more attacks of dimness. After this date the pain passed into the left upper teeth, especially the wisdom tooth. The toothache made the eye "weak," *i.e.*, dim and unable to open the lids wide; and the dimness remained permanently.

May 13*th*.—Left slight ciliary congestion; no iritic adhesions, but a number of rather large deposits on the posterior surface of the lower half of the *cornea*, not, however, arranged in a triangle; they are exactly like droplets of cold tallow; a few are confluent,

and dumb-bell shaped. Also a patch of hazy infiltration (not ulcerated) near the outer edge. *Vitreous* full of delicate films. *O.d.* very hazy, and somewhat swollen; retinal veins enlarged, and very tortuous. Numerous large, bright red, retinal hæmorrhages near *o.d.*, the largest being between it and *y.s.* at the lower part, and in contact with a large vein. No hæmorrhages at periphery. The choroidal changes consist partly of round patches, as in typical *choroiditis disseminata*, but many are irregular, and more or less linear. Most of them are unpigmented, but some show a good deal of pigment. In only very few is the choroid completely atrophic, the change in the majority being apparently confined to the retinal pigment epithelium. Some are reddish, as if caused by recent hæmorrhage. Field of vision a good deal broken. This attack threw much light on the cause of the other changes, which were in all probability due to previous attacks of hæmorrhage into the retina, and probably into the choroid as well.

Vision had at the above date already begun to improve a little, and a fortnight later (*May 27th*) was again almost as good as ever, the treament being unaltered. The corneal deposits had disappeared. Refraction was tested after complete paralysis of A. and found to be H $\frac{1}{60}$.

The *right* eye was never the subject of any attacks of dimness. There was a good deal of marginal and central haze of cornea, as if from repeated phlyctenular ulcers. V. has throughout been = 4 J. and $\frac{20}{50}$; refraction my. about $\frac{1}{16}$. Careful ophthalmoscopic examinations under atropine never showed a trace of disease in any part of the fundus, excepting the enlargement of retinal veins already described (p. 71).

May, 1877.—Has had no further relapse, and has also ceased to be liable to the attacks of temporary dimness. For several months has been losing flesh; is now very thin and pale, and looks phthisical. Has had a cough and "bronchitis," but no hæmoptysis. No family history of phthisis. No symptoms of diabetes.

The recovery of nearly perfect central vision after repeated attacks of disease leaving permanent changes in the choroid and in the pigment layer and other deeper layers of the retina, is most remarkable. The case is of great interest also from the occurrence in the same patient

of serious organic disease of the retina and choroid, and of attacks of transient dimness of sight in connection with dyspeptic symptoms. The patient's statement that these transient obscurations affected only the one eye cannot be taken as proved, particularly as the cornea of the other was cloudy, and I never myself had the opportunity of testing him during one of these attacks. The occurrence of severe toothache in connection with the last attack of retinal and choroidal disease is of much interest, although no conclusion can at present be drawn from it. The limitation of the disease to one eye, and its tendency to relapse, suggest some unilateral vaso-motor disease as the cause, aided by a condition of imperfect recovery of the diseased vessels after the first attack.

ON THE PATHOLOGY OF INCREASED TENSION OF THE GLOBE.

By W. A. Brailey, M.D.,
Curator of the Museum.

In attempting to determine the cause of increased intra-ocular pressure, I have examined, microscopically, 53 cases in which this has been at some time a symptom; its presence being indicated either by increased hardness of the globe at excision, or its former existence shown by the pushing back of the lamina cribrosa, as seen in microscopical sections of the disc.

In all these cases, some part of the iris ciliary body or choroid is affected, and it is convenient to arrange them in two great groups, in accordance with the first seat of the disease.

A. Those in which an affection of the iris has been the primary cause of the tension, the disease either remaining confined to this, as in my two cases of buphthalmos, or extending backwards so as to affect the ciliary-choroidal tract secondarily.

B. Those in which the part of the tract posterior to the iris is affected, without any obvious iritic change preceding it.

Group A includes 28 cases. In 26, the iritic affection has been due to perforating corneal ulcerations or wounds with anterior synechiæ; and in 2 cases it has depended on iritis arising probably from some constitutional cause, and resulting in the enlargement of the aqueous chamber characteristic of buphthalmos.

In all these cases there is, first, inflammation of the iris, soon accompanied by increased secretion of fluid.

The inflamed iris forms adhesions to the parts with which it lies in contact; to the lens capsule, in cases of

iritis, without prolapse; or to the cornea at the point of perforation, if the inflammation is due to a perforating wound or ulcer.

It is possible that very slight iritis has preceded many cases which, from an absence of any evidence of its prior occurrence, either from *post-mortem* appearances or from the patient's history, I have arranged under the next group.

The iritic inflammations above-mentioned are generally followed by secondary affections of the posterior part of the same tract.

In the buphthalmos cases, enlargement of the chamber of the aqueous is the most prominent symptom, and with the posterior synechia the pupil is seen to be small and immovable. There is some increase of tension of the whole globe, and a general increase of its measurements. The optic disc also exhibits the indications of increased pressure.

In the cases of iritis after corneal perforation, the cases differ a little according to the size of the area of cornea affected. If this has been small, as in a case of punctured wound, the slight prolapse of iris closes it, and we have increased depth of aqueous chamber, especially of the anterior part, with rapid slight increase of tension of the globe. But if the perforation be large, as in the case of a more extensive wound, more iris is prolapsed into it, and it may be so large that the whole iris is drawn forwards and applied to the posterior corneal surface. Inflammation of iris, with increased secretion, follows, but the extent to which this affects the size of the chamber and the tension of the globe depends on the closure of the wound. In corneal ulceration, the perforation is less, but the surrounding thinned area is greater, therefore we have more easy closing of the leak with yielding of this part of the cornea, thus constituting anterior staphyloma.

Besides the cases above-described, there are many

which would be arranged under this group, of which I see living specimens, but am not likely to have excised eyes. For example, in cases of corneal inflammation, ulceration, or slough, in which the cornea has sufficient clear area, it is often apparent that there is already some slight iritis with increased depth of the aqueous chamber. The occurrence of this increase of fluid will explain the fact that here, as will be afterwards seen, we have the nearest approach of any disease to the *post-mortem* characters of increase of tension. I have not the slightest doubt but that, by an increase of the delicacy of our methods of investigation, we should detect increased hardness of the globe in these cases, and I think that we have thus an explanation of the benefit frequently derived from paracentesis, incision of the cornea, or iridectomy.

Group B includes all cases known usually as glaucoma. In these, there is generally no history of any injury, or if the patient attributes the disease to a blow, it is a non-perforating one, and its connection with the glaucoma is often obscure. Though, as before-mentioned, I cannot altogether exclude primary iritic disease, yet the *post-mortem* appearances refer markedly to great ciliary or choroidal affection.

Of this group I have 25 specimens. In 14, the ciliary region seems the sole part involved. In five there are found patches of choroidal disease, together with the ciliary affection; and in one, the change has been principally manifested in the choroid. In this group I have also included five cases of tumour.

I have no doubt that the proportion of cases of increased tension due to choroidal disease principally, if not entirely, is much larger than the figures above given indicate, but in this class of cases the increase of tension appears to be usually gradual and moderate in amount, and therefore such cases rarely come to excision.

Of the 5 cases of tumour, 2 are choroidal and 3 retinal. In all these cases, though the choroid was impli-

cated and its tissue in places almost entirely obliterated by the tumour growth at the date of excision, yet the ciliary body and iris presented precisely the changes which will be found occurring in all cases of increased tension (see Fig. 2). Therefore, as these parts are not involved in the tumour growth, and as the glaucomatous symptoms do not appear to have come on till long after the tumour has made its appearance, I shall treat the glaucomatous symptoms as indications of an affection of the globe of just the same nature as ordinary glaucoma, and have, therefore, included them under Group B.

It will be noticed that I have considered such cases as anterior staphyloma, buphthalmos, and glaucoma to be all intimately related, only differing in their outward manifestations in the part of the tract of the iris choroid and ciliary body which may happen to be affected: and in the resistance offered to the increasing contents of the globe by its walls.

I shall proceed to discuss, *first*, such symptoms of these three diseases as may be more easily perceived before excision, and, finally, I shall treat of the *post-mortem* appearances.

The size of the *aqueous chamber* varies with the increased or diminished secretion of fluid from the iris. Whether the increase is found in the anterior or posterior part of the chamber of the aqueous, depends on the adhesions of the iris. In buphthalmos and any similar serous inflammations of the iris without perforation of the cornea, the anterior chamber is increased in size, whereas in similar inflammation of the iris occurring after its prolapse into perforating wounds which have subsequently closed, the anterior chamber is very small, or even obliterated, if the prolapse is great; while the posterior chamber is considerably increased. An exception to this occurs in one case of minute perforating wound where the surface of the iris has prolapsed only a little way into the wound and thus closed it. Here the

increased fluid is found especially in the anterior chamber. This is also the case in those cases of iritis with increased secretion that I have observed in unexcised eyes with entire corneæ.

The size of the aqueous chamber also depends somewhat on the age of the patient, and on the mobility of the lens system. The young healthy cornea allows of considerable yielding, so that it becomes enlarged in all directions, and also more convex; but in older eyes, there is greater corneal rigidity and, provided that the cornea be healthy, the main part of the increased space for the aqueous is provided for by the displacement of the lens backwards.

If such cases are examined *post-mortem*, the apices of the ciliary processes are seen stretched considerably backwards. But when the iritis is succeeded by atrophy of the iris, the aqueous is much diminished, or even altogether disappears. Then the pressure from behind reasserts itself and the lens is pushed forwards, in some cases, so much as to be adherent to the posterior surface of the atrophied iris, which, by the disappearance of the fluid in the anterior chamber, has been applied and is now adherent to the posterior corneal surface.

This pushing forwards of the lens more readily happens if, by an extension of the iritic disease to the ciliary body and choroid, there is secondary increased pressure in the part of the globe posterior to the lens. And if this region is affected before or without the iris, as in ordinary cases of glaucoma, the aqueous chamber is shallowed by the increased pressure, though not obliterated, because the iris still retains its functions.

The size of the vitreous chamber is also liable to be affected by increased pressure, but any remarks about this will more advantageously come under the section on hardness of the globe.

The increased hardness of the globe is dependent on an increase of the contents of the globe apparently by

secretion of fluid from the iris ciliary body or choroid, and so bears a close relation to the size of the aqueous or vitreous chambers, and to the amount of increased space that the walls of the globe will allow.

But as the iris is a very small secreting surface compared with the ciliary and choroidal tract, so it must affect the general hardness much less. Moreover, its inflammation is very often in connection with a thinned cornea, or one imperfectly cicatrized after perforation; by the yielding of which increased space is provided. In consequence of this, the tension of the globe is rarely increased in cases of recent corneal ulceration. And it is even diminished, for in cases of extensive ulceration I have seen minus tension with increasing size of the aqueous chamber from extremely ready yielding of the cornea.

But where the iris is affected apart from the cornea, some increase of general tension of the globe occurs, even though the inflammation has not extended backwards beyond the iris. Thus, in my cases of buphthalmos, though the ciliary region and choroid seem healthy, we have some cupping of the disc, and similarly, in many cases of serous iritis which are seen during life, we have plus tension. This cupping of the disc or increased tension, is usually slight on account of the comparative total increase of fluid being slight, and its distribution over both chambers principally by the mobility of the lens system, though there appears, also, to be some intercommunication of fluid between them. Similarly, increased hardness of the globe is connected with general or local yielding of the walls of the vitreous chamber, but it will be best treated of after we have examined the causes and extent of this.

It is obvious that if the increase of the contents of a glaucomatous eye be limited and definite, as in the cases where the inflammation is of short duration, and does not lead to much structural alteration of the parts affected, so

a slight increase of space, or the escape of some of the contents may be sufficient to afford permanent relief to the condition. This is seen in cases in which paracentesis of the cornea permanently cures increased tension, and when after slight general or local enlargement, further increase in size or hardness ceases. The same result is attained if the resulting cicatrix or staphylomatous area allows of the transudation of fluid.

This general yielding of the cornea has already been treated of, and similarly general yielding of the sclerotic is found in all young eyes, and, to a less degree, in adult eyes, in which the tension has been increased. It is seen in the cases of buphthalmos, and also in those young cases in which secondary affection of the ciliary and choroidal tracts follows iritis after perforation. The increase of measurement is seen in all directions, though if the cornea have originally been ulcerated the antero-posterior measurement is the one most affected. But if the disease has affected solely or with especial severity limited areas, then we have atrophy of the sclerotic over the parts affected. If the sclerotic be the yielding one of a child, these parts give to the pressure, and there is relief by the local bulging. As it was said that cases of increased tension are sometimes connected by the patient with non-penetrating blows, so these local thinnings occur more commonly in the parts most exposed, *i.e.*, in the upper ciliary region, though they are found to a less extent elsewhere.

Thinning of the corneal margin, independent of any previous local disease and consequent marginal staphylomata, are by no means uncommon. These, however, are, I think, due to pressure from behind, causing absorption of the posterior corneal surface, as will be treated of subsequently. These corneal marginal staphylomata are generally lined by a web of pigment, representing atrophied iris, through which the pressure is applied. But cases occur in which they are only partially so lined, or even are entirely free.

I have seen cases in which, notwithstanding that the posterior corneal tissue has been thus absorbed, there is no loss of total thickness in this region, as nucleated fibrous tissue has been added on to the exterior to an equal or even greater amount.

But in senile eyes, and particularly in hypermetropic ones, which are in especial the subjects of glaucoma, there is normally a thick and rigid sclerotic, and consequently the symptoms and consequences of increased tension are rapidly developed and very grave.

If, however, the disease be of long standing, scleral atrophy is produced here, though less speedily than in younger eyes. This atrophy may be general, and in one case, though the sclerotic was reduced all over to about $\frac{2}{5}$ths of its normal thickness, there was still plus tension and no increase of size of the eye, or it may be local, and then dark patches are seen, having the general appearance of local scleral staphylomata. Here, also, the rigidity of the part remaining tends to prevent bulging to such an extent that in one case, though the sclera at the patches was scarcely a third of its normal thickness, with still increased tension, there was no elevation of the thinned areas.

Corneal prominences, after extensive ulceration, probably owing to the altered character of the cornea surrounding the perforation, occur equally readily at all ages, though they are far more common in children, as corneal ulceration, especially such as is followed by cicatrisation of the perforated place, is far more common in them.

Other pathological glaucomatous changes are much better seen in excised eyes, though some of them are capable of recognition before excision. They may affect:—

1. The optic disc.
2. The iris.
3. The ciliary region or choroid.
4. The vascular supply of the globe.

The changes are given as found in the specimens at

my disposal, but among them, I regret to say, there are not any cases of unadvanced glaucoma simplex. Most of them are cases of glaucoma absolutum, but in some of these the disease has been acute, and tolerably recent. I have one case of those I have included under group B in which the outbreak of the disease dates from less than six weeks before excision, but with this exception I have none in which the history shows the case to be clearly of less duration than five months. There are included, however, under this head, cases of increased tension of short duration only, secondary to tumours, in one case the patient being but two months old.

Of glaucoma secondary to iritic disease I have one case seven months after the corneal ulcer had made its appearance, and another ten months after. I have several early cases, in which there is anterior staphyloma still yielding with involved iris, but no apparent increase of tension, and I have examined many times the iris removed in the iridectomy of early cases of glaucoma simplex.

With reference to the symptom of *cupped disc*, I find that in point of time it follows at some distance the increased tension, even where this has been very considerable.

For example, the cup, with five months' history of increased pressure, it being registered as + 2 at excision, is shallow, wide, but not at all undermined at its sides; and the case of less than six weeks' duration presents, with a registered tension of + 3, a deep, though not at all flask-shaped, pit. I find no case with a cup whose edges are distinctly undermined without a history of at least two years. And in many cases with a history of seven and more years, and tension + 1 at excision, the cup is neither deep nor undermined laterally.

While in order to produce a flask-shaped cup it requires a history of several years of increased tension, yet even this duration of but moderately increased pressure is not sufficient, and we have only a wide pit whose central part is usually the deepest.

Though the formation of a cup, and especially one with undermined edges, takes a long time, yet in all the cases in which increased tension has been noted before excision I have found distinct evidence of it in the disc.

The first result of the pressure is a pushing back of the centre of the lamina cribrosa, so that its front surface, instead of being nearly straight as in normal eyes, is markedly concave (Fig. 3). This happens before there is any other naked-eye indication than a small central pit, and can only be well seen in microscopical sections of the nerve entrance parallel to the course of the nerve fibres. There accompanies this altered position of the lamina cribrosa a disappearance of the nuclear tissue, among which the blood-vessels lie in the centre of the disc, so that they are pushed to the sides of a central funnel-shaped, ill-defined, small depression. This extends down as far as the lamina cribrosa, and afterwards enlarges in a lateral direction. If the pressure is never very great, the affection of the lamina cribrosa does not get greater, and further change is limited to a replacement of the nerve fibres in its concavity by nuclear tissue, and the subsequent absorption of this. So that, with only moderate increase of tension, first we have a pushing back of the centre of the lamina cribrosa, with a central pit, not extending down as far as it, then a wider funnel reaching the lamina cribrosa, and then lateral increase, till the inner scleral edge of the optic nerve entrance is almost laid bare, but there is no undermining.

If the pressure is still greater, the lamina cribrosa is pushed back along its whole extent, so that its lateral parts line the overhanging anterior scleral edge of the nerve entrance (Fig. 6). Thus we have here more rapid destruction of nerve fibres than the previous description of the ultimate results of long-continued less tension shows, though, as far as the total destruction of nerve fibres are concerned, the effects are the same.

In a well developed cup the floor is formed by the

lamina cribrosa, which is denser than usual, from its fibres being pushed together in an antero-posterior direction (Fig. 6).

General increase of size of the globe by diminishing the pressure, checks the formation of a perfect flask, and therefore in young eyes of long standing increased tension, discs are more saucer- than flask-like.

Atrophic depressions of the optic disc from absorption of its fibres are much spoken of as symptoms of some forms of disease, and do without doubt occur. But here the lamina cribrosa, though laid bare, retains its usual level. But I suspect that very many of the so-called atrophic cups as seen by the ophthalmoscope are due to very long continued subacute glaucoma simplex, in which the tension has never been or has ceased to be very palpably beyond the normal.

It is a most important and interesting question as to whether cupping of the disc is preceded by swelling. Certainly swollen discs are found in all cases of inflammation of the iris and its continuations, very closely following perforating wounds or ulcers. And therefore I have no doubt that very many cases of cupped disc are preceded by swelling. But the swelling of the disc may be due to the diminished tension, as the cupping is ultimately to the increased pressure, and so they are both secondary results of other changes, and neither is in itself a primary disease.

Swelling of the nerve entrance due to changes outside the globe may be followed by atrophic cupping.

The after-history of a cup in those old cases in which the tension has fallen either spontaneously or after operation, is a matter of great importance. I have found many such cases in which the depression has been refilled almost entirely by nuclear matter, so that to the naked eye no indications whatever of a cup remained. But microscopical sections show that the lamina cribrosa still retains its depressed condition, though perhaps somewhat diminished

from its original extent. And I believe that in all cases this alone will unmistakably indicate the former existence of increased tension, though it is out of my experience, that some other pathological glaucomatous condition is not found to give confirmation to it.

The *ciliary body* is the seat of the most marked and universal changes occurring in glaucoma. For of those cases included under Group B, it is extensively affected in all but one, and here, even, there is some change. And in those included under Group A, it is markedly implicated in all but the two buphthalmos cases, and the single case of small, rapidly-closing, perforating wound of the cornea, with anterior synechia.

The remaining 49 cases of increased tension present marked ciliary changes, and the one which is present in all, and more distinctly seen in each, than any other pathological condition, is atrophy of the ciliary muscle (Fig. 4). This atrophy is well seen in all, and almost equally in those cases of short and of long duration, provided that the increase of tension has been great in both. It affects in particular the circular fibres, and, therefore, the antero-internal part of the muscle (with relation to the antero-posterior axis of the globe) is the most wasted, and in consequence of this the muscle, instead of being of its greatest thickness in front, often tapers forwards from a point some way behind its anterior border. This affected part in many cases presents no trace of muscular fibres at all, but in most there are a few thinned ones mixed with a little nuclear tissue and some blood-vessels.

The radial fibres do not escape unaffected. Their bulk is diminished, though to a less degree than the others. And even this does not represent the full extent of the atrophy, for the fibres left are much thinned, especially those forming the central part of the muscle, and here, frequently, they can be scarcely seen at all. In some specimens this condition exists throughout both sets of

fibres, and scarcely a trace of the existence of muscular tissue can be found anywhere.

This atrophy does not find full expression in measurements of the muscular thickness of the front part, both on account of the thinning of the muscular fibres without fully corresponding general diminution of the muscular size, and also from the somewhat fusiform shape of the muscle. Moreover, in many cases, both in healthy and morbid muscles, the antero-internal border extends considerably in advance of the origin of the radial fibres (Fig. 1), and so the thickness of the muscle opposite their origin is somewhat diminished.

This last source of fallacy I have attempted to correct by halving the distance that this part of the muscle thus extends in advance, and adding the result to the muscular thickness opposite the origin of its radial part.

I think that in some cases this muscular advanced position may be due to anterior synechiæ, but in general, if I had altogether neglected it, the ciliary atrophy would have been still more markedly expressed in my figures. I have made no correction for the distance the greatest muscular thickness is behind the radial origin, though this would have still more emphasised the atrophy, but have recorded the greatest muscular diameter wherever found.

A contracting retina, which still retains its attachment at the ora serrata, may apparently increase the size of the muscle, but this disease rarely is concerned in my measurements.

In the three cases under Group A, in which the ciliary muscle is not markedly atrophied, there is in all decided increase of fluid in the aqueous chamber apparently due to disease or injury of the iris with synechia. And though the general hardness of the globe is rather increased, there is no evidence of disease affecting the ciliary and choroidal tracts beyond the iris. In the case of injury, the wound occurred only 14 days before excision, possibly too short a time for the inflammation to have travelled backwards.

In all the other cases of this Group A, increased tension has followed an entanglement of the iris, and the ciliary muscle in each case is found to be atrophied, especially as to its circular fibres, to a degree corresponding pretty nearly with the *amount* of increase of tension rather than to the time of its duration. This is extremely well seen in those specimens with increased tension after injuries, excised on account of their setting up sympathetic ophthalmitis in the other eye. In each of them the iris is found to be entangled in the corneal wound, and the atrophy specially prevails in the part of the muscle nearest this. This atrophy is very early developed, occurring in the earliest specimens in my possession. And as in cases of secondary glaucoma the tension is generally less than in Group B, so we find the average muscular size larger.

In the single case included under Group B, in which this muscular atrophy does not very markedly occur, there is evidence of choroidal disease, and I believe this to be principally the cause of the increased tension. In the cases of glaucoma accompanying tumours of the retina and choroid included in this group, the ciliary region, though unimplicated in the tumour mass in all the cases but one, is greatly atrophied. And it is remarkable to notice that this change is observed not only on that side in closest relation with the tumour, but on the side removed from it. Just as local choroidal disease has been seen to correspond with scleral thinning, so with atrophied ciliary regions of some standing we have diminished scleral thickness. If this atrophy be limited to part of the circle, and the increased pressure be great and the subject not old, relief is found by the establishment of local ciliary scleral staphylomata.

Examining microscopically specimens of other diseases, I find that in old lost shrunken eyes there is an atrophy of the ciliary muscle, but this proceeds *pari passu* with general atrophy of the structures of the globe, and does not affect especially the circular fibres, nor is the muscle

wasted in advance of any other structure. Examining other eyes with normal tension, I find that in old wounds of cornea without iritic adhesion, after iridectomy or cataract extraction there is no atrophy. Similarly, after cataracts of long standing without other affection. Nor is there any found after suppurative iritis or detached retina recent or old. And, even in adhesions of iris to the cornea or lens, independent of perforation, and probably due to iritis from some general cause, no atrophy is found.

But in opacities after corneal non-perforating ulceration, there is slight diminution in size of the muscle, and this is rather more marked in sloughs of the cornea, and the same in a case of epithelioma affecting this region.

In adhesions of the iris especially to the cornea, after wounds or perforating ulcers not followed by plus tension, the atrophy is greater than in the diseases just enumerated, though even here it is below that occurring in the mildest cases of increased tension.

It is very interesting to note that the diseases I have just enumerated as those in which the greatest atrophy occurs, short of increase of tension, are those which are especially liable to be accompanied or followed by increased hardness; and, moreover, that where slight atrophy accompanies synechiæ or ulceration, it is frequently seen to be greatest on the side corresponding to the morbid condition.

It is possible that this atrophy may be the cause of the diminished power of accommodation which prevails long after the disappearance of corneal ulcers.

My reasons then for thinking that this atrophy bears a very close relation to increased tension are—

(1.) Because it is found only markedly here.

(2.) Because the diseases in which it occurs to a slight extent, with tension which we cannot with the finger distinguish from normal, are those in which plus tension very frequently comes on later.

(3.) Because the amount of atrophy of the ciliary

muscle bears a close relation to the amount rather than the duration of the increased tension.

(4.) Because the atrophy in secondary glaucoma follows the iritic adhesion, and precedes in point of time any rise of tension that is distinctly appreciable.

In other cases during life I have seen evidence of this atrophy; in one case, diagnosed as old cyclitis with increased tension, and apparently unaffected iris, bluish patchy thinning of the ciliary region was apparent; in another with increased tension after perforation of cornea and iritic prolapse, the whole ciliary region was thin and bluish.

That this condition is by no means a result of increased pressure is made clear by two cases, one in which, acute glaucoma of less than six weeks' duration, having necessitated excision (Figs. 4 and 5), the upper part of the ciliary muscle is reduced almost to $\frac{1}{3}$ of the thickness of the unaffected lower part of the circle; and the other in which subacute glaucoma occurred in an eye from which a cataractous lens had been extracted through the pupil many years before. Here the upper limb of the iris is adherent to the scar; and the corresponding ciliary muscle is little more than two-thirds the thickness of the part below, and its fibres are thinner. In both these cases, sections of the lower part of the ciliary circle show it to be not far removed from normal measurements (Fig. 5).

Taking the 25 specimens which make up the entire Group B, I find the average thickness of the ciliary muscle to be ·361 mm., the average age of the patients 43·6 years, and the mean duration of the disease 4·8 years. If, however, the cases in which the increased tension is due to intra-ocular choroidal or retinal tumour be excluded, as being exceptional in their cause, as well as in affecting a younger class of subjects, the average muscular thickness is ·356 mm., with an average age of 50 years, and an average duration of disease of 5·8 years. But if we take the tumours alone, though we find the average muscular thick-

ness to be not very different, being ·394 mm., yet the average age is only 15 years, and the duration about 1·4 years.

In nearly all the above cases, measurements of at least two points of the ciliary circle have been taken. Similar enquiries as to four tolerably healthy eyes in my possession yield with an average muscular thickness of ·8 mm. an average age of 44 years. Taking again four diseased eyes in which no increase of tension has ever been manifested, there is found ·84 mm. for the muscular thickness, and 39·5 for the age. The mean duration of the disease is here 19·5 years, but much importance cannot be attached to this, as in one, a case of kerato-iritis, it is as long as 58 years, while in two others it is less than one year.

Examining for purposes of comparison all the 28 specimens of Group A, we have an average muscular thickness of ·435 mm., an average age of 31 years, and an average duration of disease eight years.

But if we treat by themselves the three cases in which the iris seems to be the part almost if not exclusively affected, the average size is ·68 mm., the age 30 years, and the duration of disease five years.

The question then arises, is this atrophy secondary to inflammation of the same part, or is it primary? And then, if it is secondary to inflammation, is this or the atrophy the cause of increased tension?

In the earliest cases that I have of Group B, I find rather numerous nuclei lying between the muscular fibres and in the tissue of the base of the iris. And this increase can be especially noticed by contrasting the sections of the healthy and the affected muscle where one portion alone of its circle is implicated (Figs. 4 and 5). In the cases in which the tension has been of some standing, the nuclei between the fibres of the muscle are even fewer than normal. And this will account for the fact that being without very early cases, I have none in which the nuclei are *very* abundant.

In early cases of secondary glaucoma (Group A) affecting the ciliary muscle, I find a great increase of nuclei, and this is better seen in the part of the muscle near the affected iris. Later a diminution follows, as in the cases of Group B.

But in other diseases not resulting usually in increased tension, as suppurative iritis and ophthalmitis and wounds of the cornea without synechiæ, this nuclear increase is still more marked; therefore, though increase of nuclei is in many cases a first stage in the glaucomatous process, it is by no means necessarily its cause.

I should judge that some form of inflammation resulting rapidly in atrophy, whether affecting the iris ciliary body or choroid, is the cause of increased tension.

In many cases, increased tension occurs so rapidly, that it is impossible to consider it due to atrophy. This is seen in displacements of the lens whether into the aqueous or vitreous chamber (of which several very interesting cases are given by Mr. Bowman in vol. v of these Reports) and in many cases of punctured wounds of the globe. In these cases it is far more likely that an inflammation of the same kind as iritis serosa, iritis with corneal ulcers, &c., takes place, and that this may subside, relieving the tension and leaving the muscle unaffected, but that far more commonly it continues longer, and is succeeded by more or less complete atrophy. It is possible, however, that in displacements of the lens there may be temporary or permanent loss of function of the ciliary muscle from its being dragged on. But many eyes are excised on account of glaucoma absolutum, the tension being as high as it ever was, in which all traces of inflammatory disturbance of the ciliary muscle are absent from this region and other parts of the same tract. But in these complete atrophy of the ciliary muscle exists, and it is a question in these cases as to how the tension is kept up. Exclusion of the pupil or marginal anterior synechiæ, such as are found in the vast majority of cases of Group B, and in most of Group A, are

without doubt factors in keeping up plus tension; but it is not entirely due to this, for tension with atrophy of the muscle persists in several cases, in which there has never been any peripheral adhesion, and in cases in which this has only implicated a small segment of the periphery of the iris. Moreover, cases occur in which there is peripheral adhesion without any increase of tension. Therefore we must conclude, that though inflammation of the ciliary region is very frequently a cause of increased tension, yet that this is maintained in connection with the muscular atrophy.

In Group A the inflammation and atrophy of the ciliary muscle where these occur are secondary to similar changes in the iris. In Group B, though similar iritic inflammatory and atrophic changes are found, yet is is probable that these in most or all are secondary, but of this I shall treat more fully under " Pathology of the Iris."

The part of the ciliary body on the internal aspect of the muscular fibres contains sometimes larger veins than usual, but in thickness seems not diminished. The ciliary processes are frequently twisted and somewhat atrophied.

I have but one case in which the choroid might be considered as the principal cause of the increased tension; it being in all others much less visibly affected than the ciliary body and iris. In this case the change is simply one of atrophy. I believe that cases of glaucoma due to choroidal disease are not uncommon, and I have recently seen a case in which white patches are visible to the ophthalmoscope with increased tension, but such cases I imagine rarely come to excision. In the other cases in which I have examined the choroid I find usually atrophy more or less extensive.

Changes in the *Iris* in increased tension are very marked, though not so closely connected with it in time as the ciliary atrophy; seeming to precede considerably in many cases the decided increase in Group A, while in Group B they are without doubt secondary.

These changes may affect its position and relations or its structure.

In all cases of Group A there has been iritis as a first symptom. This iritis may be primary, as the iritis which has led to buphthalmos, and in this case the adhesion of the iris is to the anterior surface of the lens capsule. In consequence of this posterior synechia, a very deep anterior chamber is visible in both my specimens, and the pupil is small, fixed, and centric, the adhesion of the pupillary margin being almost or quite universal, and being slowly succeeded by atrophy of the iris.

In the other cases of Group A after wounds and perforating ulcers, as pointed out under the general symptoms of this group, iritis with increased serous secretion either precedes or is due to the prolapse. If the perforation is small it soon becomes closed, and we have simply a point-like anterior synechia, but if large, much iris prolapses, frequently even the whole pupillary margin. Serous inflammation of the iris is set up, its periphery becomes adherent to the periphery of the cornea to which it is applied, and the remainder of the iris generally lines the cornea between the adherent base and pupillary edge, taking on the exact shape of its posterior surface, and finally becoming adherent there. Atrophy rapidly succeeds and finally no trace of iris is left but a web of pigment closely adherent to the posterior corneal surface. The anterior lens surface, at first far removed from the iris by increased fluid in the posterior chamber, is pushed forward as the iris atrophies, and finally becomes adherent to the web of pigment.

In the subacute and aphakic cases of Group B, the iris is free from synechiæ, as it also probably is in all *early* cases of glaucoma of this group, so that it remains free and mobile and is unatrophied. But in the great majority of my specimens its periphery is bulged forwards and outwards so as to line the rim of the anterior chamber of the aqueous (the space of Fontana) (Figs. 2 and 4). It is ap-

plied for a little way to the posterior surface of the periphery of the cornea, but again becomes free to the pupil. This condition of things may exist for some time, the pupil being dilated and only imperfectly mobile owing to this position of the iris margin, and centric or eccentric according as the adhesion exists only on one side or equally all round. Now if the fluid be allowed to escape from the aqueous chamber, as in paracentesis, the iris will regain its normal position and mobility.

But more frequently the applied surfaces become permanently united owing to inflammation, as evidenced by increase of the nuclei of the iris affecting especially its base and anterior surface.

The adherent part rather rapidly atrophies and sometimes is reduced to a mere web of black pigment on this part of the cornea. The free limb also atrophies but vastly more slowly, and the anterior surface, from its previous inflammation, contracts and rolls forward the part of posterior surface nearest the pupil, so that its pigment presents at the pupillary edge and is seen in front. In one specimen which I have the iris is thus rolled on itself like a scroll, and was not at visible all during life.

In this group, B, the inflammation of the base and anterior surface is probably secondary in time to the ciliary disease, which is here the principal symptom; at all events, it is distinctly so in one case in which the iris lines the rim of the chamber, but is neither inflamed nor adherent. But I will not say that it never is primary, and thus the starting point of the glaucoma.

This peripheral adhesion of the iris is extremely common, occurring in all my cases of Group A, except the buphthalmos cases and the case with small anterior synechia after a wound, and in Group B in all but three cases, though in two more, only one part of the iris is thus adherent and the rest free; while in another, one part, though not yet adherent, lines the rim of the chamber.

The atrophy of the adherent periphery always suc-

ceeds, though it varies very much in degree, according to the length of time that the adhesion has existed. It is very slowly followed by atrophy of the free limb.

It has been before noticed that inflammation of the iris causes increased aqueous secretion and its atrophy a diminution of the fluid. This last condition, together with the increased pressure from behind, is the cause of the shallowness of the aqueous chamber which occurs in most cases of glaucoma.

This peripheral adhesion of the iris, though found in 25 out of 28 cases in Group A and in 22 out of 25 cases in Group B, is not the cause of the increase of tension, as has been maintained by Dr. Max Knies in a very able paper, both from the buphthalmos cases quoted above and the other case of Group A with increased size of anterior chamber; for in all these the margin of the iris is widely removed from the cornea, and also from a case in which universal peripheral corneal adhesion of the iris is found after old kerato-iritis, there being no evidence of plus tension in the disc, and from another case in which a marginal corneal wound has caused a corresponding local peripheral adhesion without increase of pressure, and from yet another in which permanent peripheral adhesion, after corneal perforating ulcer, was followed by an extremely slight increase of tension, which subsided spontaneously.

Notwithstanding all this, these adhesions have, without doubt, great effect in keeping up an already established increased pressure, for in the examination of four glaucomatous eyes, in three of which iridectomy and in one iridectomy and subsequent sclerotomy have been performed without relief of the tension, I find that, with but one exception, the periphery of the iris has been left as a more or less atrophied layer still adherent to the cornea. In all these three cases with adhesion left, the ciliary muscle is much atrophied, but in the other one there is much inflammatory disturbance, but also a diminution in size of the ciliary muscle. In this case, notwithstand-

ing the removal of the iris right up to the periphery, the increased tension has returned.

I have moreover observed many cases during life in which plus tension has existed with a centric and mobile pupil; the iris when examined after iridectomy showing no signs of atrophy, in which iridectomy right up to the margin failed to produce relief of tension; as well as many others in which the margin was left adherent, with the tension unrelieved.

From the cases cited, I am inclined to the belief that if the affection of the ciliary body or choroid has run on to atrophy, then iridectomy will permanently relieve the hardness, but that if the increased nuclear condition still persists it will fail to give relief.

The cause of this application of the periphery of the iris to the rim of the aqueous chamber seems to be an increased flow of fluid from behind the plane of the lens, issuing somewhere between the ciliary muscle and the periphery of the lens, for as before said, in aphakic cases, where the communication is widely opened, and in subacute cases where the flow is slight, this condition does not exist. It is clear that the cause is a mechanical one from the case in which the iris is applied, but non-adherent; and I do not think that the advance of the lens will account for it.

That the iris seems not to allow the passage of fluid through it, from its posterior to its anterior surface, seems to derive confirmation from the fact that when the iris prolapses into a corneal perforating ulcer or wound, this ceases to leak if the iris is large enough to fill it completely, and we have anterior staphyloma following; and also from the bulging forwards of the iris between its pupillary edge and its periphery, frequently seen with exclusion of the pupil. In the same way I think the increased pressure bulges the iris forwards in glaucoma.

Being thus applied or united to the periphery of the cornea, it will prevent the transudation of fluid in this

region, and as the conjunctiva here leaves the corneo-scleral junction uncovered, this seems the most likely place for the escape of fluid. Moreover, this part of the cornea is most subject to staphyloma, independent of wounds or ulcerations; and though this often occurs with adhesion to it and atrophy of the iris, yet it sometimes occurs where the iris is entirely or partially free, and the cornea therefore unlined at least in part by it, thus leading to the supposition thut there may be increased pressure with consequent greater escape of fluid here than elsewhere.

Therefore marginal synechiæ will, without being able to cause glaucoma, yet tend to keep it up when once established by diminishing the escape of fluid; and though the way in which these adhesions act may be a matter of speculation, yet the results of their action are clear matters of fact. This peripheral adhesion can be frequently made out in glaucoma cases during life, and the anterior chamber is generally a trifle limited and shallower on that side, but very noticeably so if the case is an advanced one. I have several sections showing the condition only just being established, for the base of the iris is adherent to the ligamentum pectinatum iridis, but has not attained to the cornea.

In *every* specimen of Group A iritic adhesion is found, and in 26 out of the 29 cases this adhesion affects, though rarely is limited to, the periphery of the chamber.

In 22 out of the 25 cases of Group B peripheral iritic adhesions are found, and in the remaining three the iris is entirely free.

What I have to say about pathological changes in the *blood-supply* refers only to the condition of the canal of Schlemm and the veins in the ciliary muscle.

Schlemm's canal is commonly closed in my specimens of increased tension by apposition of its internal and external walls, but this is by no means universally the case, for this closure is a much less constant condition than either the atrophy of the ciliary muscle or the atrophy or adhesion

of the iris. In fact in some cases, notably the two specimens of buphthalmos, a very patent condition of Schlemm's canal is met with; and therefore I am disposed to attribute its closure to its compression, either through the dragging forward of the iris in a prolapse or its bulging forwards from pressure behind, as in the cases of Group B. And comparison of the cases in which the closure occurs supports rather this view. In these cases also the nuclei in the tissue surrounding it are increased somewhat, but this, like the closure, is so variable a symptom, that I do not attach much importance to either as causes of increased tension. I rather look on them both as results: the former of increased pressure, and the latter of contiguity of the inflamed base and anterior surface of the iris. Considerable importance has been attached by Dr. Max Knies in his very excellent paper to this condition, but I think wrongly.

In studying longitudinal sections of the atrophied ciliary muscles of glaucomatous eyes, it is very common to find sections of dilated empty venous channels. These are of more frequent occurrence than the changes in Schlemm's canal, but are not so constant as the ciliary and iritic changes above described. I am unable to say precisely what may be the pathological causes or results of this condition, but the atrophy of the muscle may be connected with yielding of the muscular tissue surrounding its veins. The increased intraocular pressure should have a tendency to close these veins. We may connect them with the dilated ciliary veins so often seen externally during life in such cases.

It is extremely likely that the ciliary muscle has functions besides that of accommodation, and that it may be concerned in regulating the blood supply. This might explain the effect of an atrophied ciliary muscle in passively keeping up an increased tension, notwithstanding that the periphery of the iris has never been united to the cornea.

Certainly the existence of fairly-developed muscles in eyes which have been for long cataractous, (in one of my cases for 15 years, and in one from which the opaque lens had been removed for 11 years), and in many very senile cases, seems to show that its functions extend beyond that of accommodating the eye for near vision.

But if the ciliary muscle have these functions, its paralysis should be accompanied by increased tension.

This, however, tends to be the case, for in glaucoma one of the earliest symptoms is the paralysis of accommodation; and primary glaucoma (Group B) is far most common in senile eyes, where the accommodation is feeble, and in hypermetropic senile eyes where the strain upon it is especially great. Moreover, I have seen several well-marked cases in which, with slight increase of tension, the use of atropine has caused a great exacerbation of the symptoms.

I have no personal experience of any value of the effect of atropine on the tension of healthy eyes, but that it does not cause any unmistakable increase of tension need not, considering the double course of the muscular fibres and possible double function of the muscle, much affect my argument.

For more particular information as to its effect on the tension of diseased eyes I must refer to the experience of others.

From the previous observations I draw the conclusion that, as increased tension is for long unaccompanied by any ophthalmoscopic change in the optic disc, and as moderate long-continued tension will not produce a cup with undermined edges, the term *glaucoma cup*, as applied to this form only, should be abolished.

If I may venture to derive from pathology a few suggestions for operative treatment, I shall draw attention to the fact that even where the peripheral marginal corneal adhesion of the iris exists only at one part of its circle, an iridectomy here, which must necessarily leave the peri-

phery of the iris unfreed from the cornea, will not relieve the tension; and therefore I should choose that part where the iris is unadherent.

The danger of leaving iritic adhesions after operations is very well known, but the way in which they act, by causing iritis and finally inflammation and atrophy of the ciliary muscle, with, I believe, consequent increased tension, is well illustrated in some of my specimens.

DESCRIPTION OF THE FIGURES.

Fig. 1.—Antero-posterior section in a horizontal plane of inner ciliary region of Alfred R., æt. 10, a patient of Mr. Couper. (No. 110.)

Keratitis from contact of lime 6 weeks before excision. Conjunctiva destroyed. Tension normal at excision, no evidence of increase at any time.

Muscular fibres well developed, both longitudinal and circular; thickness opposite origin of radial fibres = ·71 mm.; most anterior part of muscle extends ·34 mm. in front of the radial origin.

Iris quite free; thickness about = ·25. Its nuclei and those of ciliary body are increased.

Corneal and scleral tissue infiltrated with nuclei. Epithelium wanting.

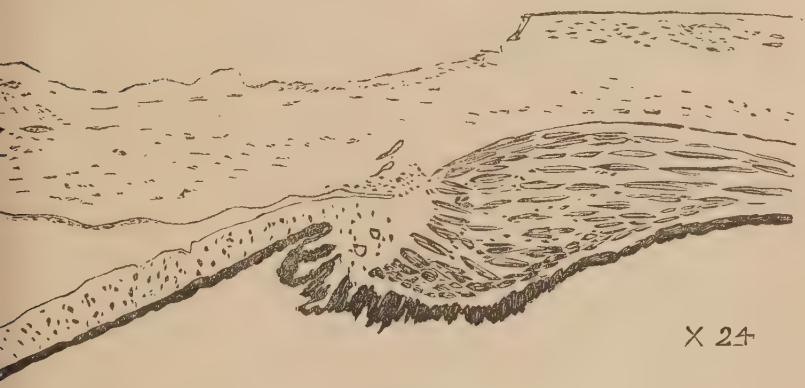

Fig. 1.

Fig. 2.—Antero-posterior section in horizontal plane of *inner* ciliary region of left eye of Lucina G., æt. 29, a patient of Mr. Hulke. (No. 121.)

There was a small sarcoma of the choroid at the *outer* side, extending

from 2 mm. in front of optic disc forwards, to within 5 mm. of ora serrata. She first noticed the eye dim 2 years ago. At excision tension = + 1.

Ciliary muscle has a maximum thickness of ·42 mm.; muscular fibres obscurely seen; nuclei of ciliary body increased. Iris not atrophied, thickness about = ·3; periphery just lines space of Fontana; its nuclei increased.

Fig. 2.

Fig. 3.—Optic disc of right eye of Harry M., æt. 7½ months, a patient of Mr. Lawson. (No. 52.) Eye excised for anterior staphyloma of cornea after perforating ulcer, in consequence of ophthalmia neonatorum.

Tension slightly increased. There is central depression of optic disc, and a pushing back of the lamina cribrosa.

Fig. 3.

Fig. 4.—Antero-posterior vertical section of superior ciliary region of right eye of Amelia D., æt. 50, a patient of Mr. Lawson. (No. 777.) No operation had ever been performed on this eye. Glaucoma absolutum

Fig 4.

× 24.

Fig. 5.

following a blow six weeks ago ; for the last three weeks there has been great pain. *At excision* the tension is + 3.

Ten years ago she found accidentally that this eye had only perception of light. She came here and cataract was diagnosed, and an operation was advised. She, however, refused. Till the blow six weeks ago, she never had any trouble with it. Pupil dilated ; *lower* limb of iris shorter.

In this specimen the ciliary muscle exhibits very little muscular fibre remaining. Its greatest thickness is ·29 mm. Iris thin but long ; average thickness of limb = ·12 mm. ; its free end = ·21 mm. ; its base is applied and adherent to the cornea at the periphery of the aqueous chamber. Nuclei of iris and those between the fibres of ciliary muscle are a little in excess.

Fig. 5.—Corresponding section from lower ciliary region of same eye. (No. 777.)

Ciliary muscle much larger ; greatest thickness = ·7 mm. Its fibres better developed. Iris presents no adhesions ; thickness about ·3 mm. Slight excess of nuclei in both iris and ciliary body.

Fig. 6.

Fig. 6.—Optic disc of right eye of John C., æt. 44, a patient of Mr. Hutchinson. (No. 35.)

Absolute glaucoma. Disease of 3½ years' standing. Periphery of iris lines space of Fontana. Ciliary muscle much atrophied. The cup is seen to be very deep, and its edges are undermined. The fibres of the lamina cribrosa are bulged far back, and closely compressed.

REPORT ON TWO CASES OF RARE TUMOUR OF THE EYE.

By W. A. Brailey, M.D.,
Curator of the Museum.

Epithelioma of Conjunctiva.

Case 1.—*Epithelioma of Conjunctiva, involving Corneal Epithelium, of two years' duration in Right Eye of Man æt. 60—Repeatedly shaved off and cauterized at intervals of about six weeks—Return of growth and increase of pain—Excision of Globe.*

[Clinical notes by Dr. Gunn, House Surgeon to the Hospital.]

Joseph B., æt. 60, admitted under the care of Mr. Hulke on April 29, 1876. He is an active, well-nourished man, in good general health.

Eighteen months ago he first observed something unusual in the right eye. On the outer third of the corneal periphery and involving the adjacent conjunctiva is a patch consisting of a number of small red papillary elevations. It has caused slight uneasiness from the rough surface rubbing against the lid.

The epithelial growth was now shaved off with a scalpel, and the raw surface touched with solid nitrate of silver. A cold compress was applied, and gutt. atrop. and oleum ricini dropped occasionally into the eye. Discharged on May 6.

On June 14, the growth, having returned, was again shaved off, and he was discharged next day. On July 5 it seems to have been more or less subdivided into three elevated patches. These were now all shaved off and the surfaces cauterized. Some irritation followed. Discharged July 8. On November 18 there were two patches of papillary elevations, which were treated as above. This was followed by considerable pain and œdema of lids: the sloughs separated on the 2nd day. Discharged November 22. After this operation the ulceration following the slough showed no tendency to heal, and the growth seemed progressing more rapidly than before. There was now much pain, and on

December 30 the eye was excised. The left eye was quite unaffected throughout. Discharged January 3, 1877.

Sections through the growth near its thin edge show it to begin 4·5 mm. in front of the sclero-corneal junction as a thickening of the conjunctival epithelium, whose free surface rises at the anterior tumour border by a rapid slope till the epithelium has attained a thickness of ·36 mm., being more than ten times that of the healthy part near.

Here the columnar cells of the deepest row look quite normal retaining their level and resting on Bowman's membrane. The rounded cells superficial to these are much crowded. They and the still superficial squamous cells are somewhat enlarged and increased in number, but there is no marked distinction from normal in the microscopical appearance of the individual cells. The superficial lamellæ of the proper corneal tissue contain an increased number of nuclei, but all the rest of the cornea is otherwise quite healthy looking. The entire thickness posterior to Bowman's membrane is ·7 mm.

Nearer to the sclero-corneal junction the conjunctival epithelium presents the same increase of thickness, but its deep surface unlimited by any visible membrane of Bowman, projects in very short rounded papilliform cellular masses into the proper corneal tissue. Still posterior to this the thickening of the epithelium comes to an end, and the ocular conjunctiva of its usual appearance rises from the globe to be continuous with the palpebral layers. Sections through the centre of the growth show the measurement from one corneal surface to the other to be increased from 1·06 to 1·5 mm. The deepest part, with a thickness of ·45 mm. is made of corneal lamellar tissue, having rather more nuclei than normal, and bearing Descemet's membrane and its epithelium.

The most superficial part is still made of conjunctival epithelium of thickness and appearance as before described, but the papilliform projections from it are much enlarged, and extend down as broad, short columns into the superficial part of the proper corneal tissue. This is ·7 mm. thick. Its superficial $\frac{1}{8}$th has so many nuclei, that corneal tissue can scarcely be traced; but its deeper $\frac{7}{8}$ths is made of distinctly recognizable lamellæ, with moderately increased nuclei.

The cellular columns project down for a short distance, then they end by giving off several similar root-like branches. These

after a longer or shorter vertical course end by running horizontally between and separating the lamellæ, and finally terminate as gland-like rounded cell columns.

On section they are solid, and are frequently shrunk so as not to fill up the spaces in which they lie, and sometimes these are left quite empty.

The cells of the columns are just like spherical epithelial cells, only slightly larger. All the cells and nuclei in the specimen stain well.

In another section, where the tumour extends back beyond the cornea, the conjunctival epithelium, where it is only loosely connected with the globe, is affected in the same way, and its processes extend through the loose subjunctival tissue to enter the superficial part of the sclerotic.

The tumour, then, is a multiplication of the conjunctival epithelial cells. The proper tissue of the cornea is only affected as to its structure by an increase of its nuclei, though the lamellæ are separated by cell processes ramifying between them. By this means the corneal thickness is of nearly twice its normal measurements.

Case 2.—*Fibrous Tumour of the Optic Nerve.*

Sarah D., æt. 62, married; charwoman; attended the Gray's Inn Road Ophthalmic Hospital on August 22, 1876, under care of Mr. Abbott.

History, from Mr. Abbott's notes.—The patient noticed 2 years ago a protrusion of the left eye, which she attributed to a blow of a fist which she had received on the eye 6 months previously. There has been but little pain or discomfort then or since. For the last 12 months there has been no perception of light. Movements of globe limited in all directions. Some soft tumour mass can be felt behind the globe on the inner side. The optic disc is swollen, and its veins are large and tortuous.

On October 4th Mr. Abbott removed the globe, together with as much of the tumour mass as he could obtain. This seemed to be a thickening of the optic nerve to a diameter of about 20 mm., and this extended, though in a less degree, quite into the optic foramen. On cutting it through there was much bleeding. Next day the orbit was much swollen, as if from blood clots.

November 14. The swelling has gone down, but there is some supraorbital pain on the left side.

December 12. The pain still persists. The orbit is fuller than it was, possibly from a return of the growth. She is leaving London in order to live in the country.

The patient has several times since been enquired about by Mr. Abbott, but all clue to her whereabouts has unfortunately been lost.

Examination of the eye a ter excision.

The globe seems normal, except that the choroid is a little congested, and there is atrophy of the retinal nerve-fibre layer. The disc also is swollen. The optic nerve in its sheaths passes back from its entrance into the globe for a distance of about 4 mm. with a total diameter of about 4·5 mm.; it then enlarges very abruptly, and in a distance of about 12 mm. more it has swollen into a mass of about 20 mm. diameter. This is invested by a tough capsule ·33 mm. thick, which is continuous nearer the eye with the outer unaffected nerve-sheath, this having a thickness of ·16 mm. only.

Longitudinal sections show that though the intersheath tissue is thickened from a diameter of 1 mm. near the optic disc, to 2·5 mm. in the tumour, yet the greater part of the increase in size is within the inner sheath, and is due not to a multiplication of the nerve elements—for, indeed, these are totally atrophied—but to a great augmentation of the fibrous tissue normally surrounding and isolating the nerve bundles.

Near the optic disc this fibrous tissue is somewhat increased, though the total diameter of the nerve itself is somewhat less than normal. Here the nerve bundles near one part of the circumference are still visible, while at another part their spaces are occupied by yellowish, granular matter, and in the central nerve region they are in all the sections quite empty and of diminished size, though of normal shape.

The intersheath tissue is here thicker, and its fibres much more closely arranged than normal, and this extends right up to the globe in sections as a tapering column, being more pronounced on one side of the section than the other.

As this intersheath tissue passes on the tumour, so its fibres become swollen and contorted, and appear on transverse section like structureless globes, on the exterior of which are

seen numerous small nuclei. On the tumour itself this hyaline appearance is lost, and it has the appearance of a slightly spongy network of fibres, these being marked by faint parallel, longitudinal striations, and exhibiting nuclei. A condensation of this tissue forms a band representing the continuation of the inner nerve-sheath, and within this again a similar fibrous nucleated network, though more open, represents the increased fibrous tissue of the nerve. Sufficiently thin transverse sections show the spaces to be tubular, being evidently the channels of the nerve-bundles. Blood-vessels ramify along the bundles of the tumour and thick-walled blood-vessels are numerous just within the inner sheath both in the nerve and the tumour mass.

NOTES OF HOSPITAL IN-PATIENTS.

By R. Marcus Gunn, M.A., M.B.,
House Surgeon, Moorfields.

It is proposed in the following report to give a short account of each case treated in the hospital wards. The important cases will be described more at length. The present report embraces only the cases of conjunctival and corneal disease which have been under treatment since the beginning of last November; but it is intended to report the other diseases and the operation cases in subsequent numbers.

Granular Lids.

All those under treatment (13) had more or less pannus: in the majority (8) the cornea was ulcerated. Of these 10 were males, at an average age of 30 years. The remaining 3, females, had an average age of 18.

Nitrate of silver was the local application used in 8 cases. The duration of their stay in hospital varied from two to seven weeks. In the remaining 5 cases peritomy was thought necessary.

Case 1.—(Mr. Critchett.) G. L.,* pannus, and ulceration of corneæ in boy, æt. 9. Iridectomy inwards in each. A fortnight later peritomy done in left. Slight improvement. 3 weeks.

Case 2.—(Mr. Critchett.) G. L. and extensive pannus in both eyes of boy, æt. 16. Left cornea ulcerated. Peritomy in both. A month later was slightly improved: seton in each temple. (Brushed with sol. arg. nitratis, gr. x ad ℥i every second day.) Very tedious case. 7 weeks.

Case 3.—(Mr. Lawson.) G. L. and severe pannus in man, æt. 43. Left cornea very vascular. Peritomy in left. Much purulent

* Abbreviations, P. L. = perception of light; G. L. = granular lids.

discharge. (Lot. aluminis.) Cornea remained cloudy and soft. 3 weeks.

Case 4.—(Mr. Critchett.) G. L., corneæ vascular and pitted in man, æt. 29. 5 years' duration. Eyes inflamed in spring and autumn. Peritomy in both, followed by improvement in vision. 2 weeks.

Case 5.—(Mr. Critchett.) G. L., considerable pannus and partial trichiasis in both eyes of young woman, æt. 21. (Arg. nitrate in solution and as mitigated solid used to lids; gutt. cupr. sulphat., followed by gutt. zinci sulphat.) Peritomy on both eyes, and Burow's operation on both upper lids, with intervals between the operations. Much improvement. Six months later R. V. = $\frac{20}{70}$; L. V. = $\frac{20}{100}$. Reads Jaeger 1.

Five cases were under treatment for the entropion resulting from old granular lids. In two the ordinary operations for removal of lashes and excision of portions of skin and muscle were performed with good result; the remaining three were as follows:—

Case 6.—(Mr. Critchett.) Entropion and pannus in Irish woman, æt. 40, worse in left eye. Old corneal ulceration. Left eye, excision of lashes of upper lid, followed by Burow's operation. Right eye, partial excision of lashes. Result good.

Case 7.—(Mr. Bowman.) Entropion and corneal opacity in both eyes of man, æt. 54. Left eye, old perforating ulcer; anterior synechia. Burow's operation and canaliculi slit in both. Corelysis (Bowman's) and iridectomy outwards in left eye. Result satisfactory.

Case 8.—(Mr. Critchett.) Trichiasis and pannus in right eye of boy, æt. 14. Burow's operation. Good result.

Ulceration of Cornea.

The following cases were nearly all of a more than ordinarily severe type. Nos. 1 to 37 are examples of onyx or ulceration with much infiltration and hypopyon. Next follow (38—43) six cases where the ulceration had a sloughing character. Cases 44—54 are of various kinds, but all less severe than the foregoing. Those remaining (Nos. 55—69) had a phlyctenular origin.

In all, excepting six cases, the final result was satisfactory, *i.e.*, useful sight was regained. In one case (42) the eye was lost, and was excised on account of pain and sympathetic irritation.

The treatment varied necessarily according to the form of the disease, and also, to some extent, according to the experience of the surgeon under whose care the case happened to be. The yellow ointment (10 to 15 grains yellow oxide of mercury to the ounce) was found especially useful in the milder forms of ulceration, such as the majority of phlyctenular cases. The results of hot belladonna fomentation (60 grains to the pint), used every half-hour and for several minutes at a time, were particularly brilliant in cases of ulceration with hopopyon. In cases of recent onyx with hypopyon much benefit was derived from cutting through the infiltrated corneal tissue. The hypopyon was generally evacuated at the time, and seldom reappeared. In none of these cases was the corneal wound reopened, as in Saemisch's operation. Where there was much pain accompanied by increased tension, paracentesis gave temporary relief, but the most lasting benefit followed in such cases from iridectomy. Quinine internally, in doses of one or two grains thrice daily, seemed to have a most beneficial action in one or two cases of hypopyon, the pus disappearing more quickly during its administration, the other treatment remaining the same.

Case 1.—(Mr. Hulke.) Right eye: Small onyx with hypopyon in man, æt. 64. Considerable pain. (Fot. bellad. frequently.) Rapid improvement. Disappearance of hypopyon on sixth day.

Case 2.—(Mr. Streatfeild.) Right eye: Large central onyx in stout flabby girl, æt. 2. Much photophobia. Incision into onyx through superficial corneal layers. (Fot. bellad., atropine, and quinine.) Extension of onyx and paracentesis corneæ (above) four days later. At discharge, three weeks afterwards: Anterior synechiæ. Cornea comparatively clear, except small yellow projection in centre. No pain.

Case 3.—(Mr. Couper.) Right eye: Central ulcer with surrounding infiltration of cornea in anæmic boy, æt. 8. Pain; slight photophobia. Corneal section through ulcer. (Fot. bellad., syr. ferr. iodid., quinine.) Rapid disappearance of infiltration and relief of pain. Ulcer healed slowly. Slight relapse 3 months afterwards.

Case 4.—(Mr. Wordsworth.) Left eye: Small central corneal ulcer with hypopyon in boy, æt. 14. Photophobia. (Fot. bellad. and quinine.) Improvement slow, but steady; hypopyon disappeared on 19th day. He had phlyctenular ulcers on right cornea. These healed slowly under belladonna and yellow ointment.

Case 5.—(Mr. Couper.) Right eye: Central corneal ulcer with surrounding infiltration and hypopyon in man, æt. 62. Ciliary injection; severe pain. (Fot. bellad., quinine, blister to right temple.) Pain persistent, causing sleeplessness. Tension increased. Corneal section through ulcer on 14th day; slight temporary relief. Iridectomy (inwards) ten days later, followed by rapid improvement. Cornea clearing. Less pain and ciliary injection, and disappearance of hopopyon.

Case 6.—(Mr. Wordsworth.) Left eye: Ulcer of cornea with large hypopyon in delicate girl, æt. 4. Much photophobia and pain. (Fot. bellad., gutt. atrop., quinine, syr. ferr. iodid., and ol. morrh.) Progress slow. Ulcer perforated on 14th day, followed by rapid improvement and relief from pain. Restoration of the anterior chamber and no return of hypopyon.

Case 7.—(Mr. Bowman.) Left eye: Nearly central onyx, superficially ulcerated with surrounding infiltration. Right eye: Deep perforating corneal ulcer. Pain. Photophobia. Florid complexioned girl, æt. 16. Amenorrhœa. (Fot. bellad., quinine, syr. ferr. iodid., pil. al. et ferri.) Progress slow. Perforation in right on 14th day, followed by more rapid improvement. Both much benefited by calomel insufflation ten days later. Ultimately good result.

Case 8.—(Mr. Streatfeild.) Left eye: Corneal ulcer with infiltration, slight ciliary injection, and pain in girl, æt. 18. Amenorrhœa. (Fot. bellad., gutt. atrop., syr. ferr. iodid., ol. morrh., and pil. al. et ferri.) Feverishness and slight tonsillitis (?) from atropine (it had been used for ten days). Eye slightly improved Went home on account of sore throat.

Case 9.—(Mr. Hutchinson.) Left eye: Ulcer of cornea below, with infiltration, and slight hypopyon in girl, æt. 6. Photophobia. (Fot. bellad., syr. ferr. iodid., ol. morrh., liq. cinchon.) Hypopyon disappeared on second day. Ulcer healed slowly (5 weeks). A small phlyctenule appeared in right eye; soon cured by yellow ointment.

Case 10.—(Mr. Streatfeild.) Left eye: Deep corneal ulcer with hypopyon in boy, æt. 7. Slight pain. (Fot. bellad., atropine, quinine.) Rapid disappearance of hypopyon and relief from pain.

Case 11.—(Mr. Hulke.) Right eye: Onyx with thick hypopyon in pale thin man, æt. 40. No pain. (Fot. bellad., atropine, quinine, wine, and extra diet.) Paracentesis twice at interval of two days, followed by temporary improvement. Ultimate recovery slow.

Case 12.—(Mr. Hutchinson.) Right eye: Large corneal ulcer with considerable thick hypopyon in man, æt. 61. Similarly affected two years ago. Slight pain. (Fot. bellad., atrop., quinine.) Steady improvement, with disappearance of hypopyon on 16th day.

Case 13.—(Mr. Lawson.) Right eye: Three small corneal ulcers, with thick hypopyon, in delicate man, æt. 41. Slight pain. (Fot. bellad., atropine, quinine, and extra diet.) Corneal section through two of the ulcers on fourth day, with evacuation of pus. Hypopyon re-formed three days later. Sent to the country.

Case 14.—(Mr. Couper.) Left eye: Onyx with hypopyon in boy, æt. 1 year 8 months. Began 8 months ago. Right eye: Phlyctenular ophthalmia. Eczema of face. Corneal section in left. (Fot. bellad., liq. arsenicalis.) Rapid improvement in left.

Case 15.—(Mr. Hulke.) Right eye: Large central onyx with hypopyon of ten days' duration in man, æt. 39. Ciliary injection and chemosis. Slight pain. (Fot. bellad., atropine, quinine, empl. lytt. to temple.) Perforation of cornea ten days later, with relief of pain. Slow improvement, but only bare perception of light.

Case 16.—(Mr. Couper.) Left eye: Onyx with hypopyon in girl, æt. 5. Corneal section. (Fot. bellad., quinine, empl. lytt.) Rapid improvement, with disappearance of hypopyon on fourth day.

Case 17.—(Mr. Streatfeild.) Left eye: Corneal ulcer, with

hypopyon of one month's duration in man, æt. 61. "Similarly inflamed 11 years ago." (Fot. bellad., atrop.) Rapid improvement, with disappearance of hypopyon on sixth day.

Case 18.—(Mr. Couper.) Right eye: Extensive onyx with hypopyon in girl, æt. 2. Corneal section through onyx. (Fot. bellad., atropine, quinine.) No return of hypopyon. Steady improvement.

Case 19.—(Mr. Streatfeild.) Left eye: Large, nearly central onyx, with hypopyon, in woman, æt. 45. Aqueous muddy. Headache. (Fot. bellad., atropine, quinine.) On third day hypopyon rather greater. (Spirit lotion and quinine.) A week later condition unaltered. Paracentesis, followed by steady improvement.

Case 20.—(Mr. Streatfeild.) Left eye: Extensive onyx (? with hypopyon) in woman, æt. 50. Corneal section almost across entire cornea. (Fot. bellad., atropine, quinine, extra diet.) Onyx disappeared on about seventh day. Anterior chamber reformed a few days later. Dense leucoma remained with transparent line corresponding to incision.

Case 21.—(Mr. Wordsworth.) Left eye: Onyx with infiltration in woman, æt. 33. Severe pain occasionally. (Fot. bellad., atropine, quinine.) Improved steadily.

Case 22.—(Mr. Streatfeild.) Left eye: Ulcer of cornea with slight hypopyon and conjunctival injection in girl, æt. 10. Iridectomy upwards: escape of pus. (Fot. bellad., atropine, quinine.) On second day hypopyon re-formed; catarrhal ophthalmia in right. Next day thick hypopyon = $\frac{1}{3}$ in left. Two days later hypopyon nearly gone, but increased again after a similar interval. Corneal section now made. Pus escaped and was not reformed. (Syr. ferr. iodid., ol. morrh.) Wound gaped; anterior chamber formed slowly.

Case 23.—(Mr. Streatfeild.) Right eye: Corneal ulcers (two), with hypopyon, in girl, æt. 5. No pain. (Fot. bellad., atropine, quinine.) Rapid improvement.

Case 24.—(Mr. Wordsworth.) Left eye: Central corneal ulcer with hypopyon in boy, æt. $3\frac{1}{2}$. Sclerotic injection. Occasionally severe pain. (Fot. bellad., atropine, quinine.) Rapid improvement, with disappearance of hypopyon.

Case 25.—(Mr. Lawson.) Right eye: Central corneal ulcer with hypopyon in delicate woman, æt. 50. Eye has always been

"weak." Posterior synechiæ. Subject to rheumatism. (Fot. bellad., atropine, quinine.) Disappearance of hypopyon on third day. Cornea cleared rapidly.

Case 26.—(Mr. Hutchinson.) Left eye: Nearly central ulcer with fluid hypopyon in man, æt. 34, result of abrasion seven days ago. Pain slight. (Fot. bellad.) Small iridectomy downwards three days later; part of pus adhered to iris. (Spirit lotion 1 to 3.) Hypopyon quite gone next day. Cornea cleared rapidly.

Case 27.—(Mr. Soelberg Wells.) Large, deep, crescentic ulcer over middle of left cornea with thick hypopyon in healthy-looking girl, æt. 17. Fleshy corneal vascularity above: lids granular. (Fot. bellad., atropine, lids occasionally brushed with sol. arg. nitrat. gr. x ad ʒi.) Corneal vascularity increased for first fortnight, spreading round the cornea. Afterwards slow improvement, cornea becoming clear.

Case 28.—(Mr. Streatfeild.) Small, deep, transparent, central ulcer of left cornea, with slow iritis and slight hypopyon in robust looking woman, æt. 23. Much ciliary and conjunctival injection. History of recent detached retina? Amenorrhœa. (Fot. bellad., atropine, pil. al. et. ferri, 8 mustard pediluvia.) Anterior chamber leaked and was again restored several times during first three weeks. After about 14 days menses appeared. Two fine long posterior synechiæ detached by ivory hook. Eye slowly improved.

Case 29.—(Mr. Hutchinson.) Central corneal ulcer with surrounding infiltration and hypopyon of 7 days' duration in left eye of girl, æt. 7. Ciliary injection. Corneal nebula in right eye. (Fot. bellad., empl. lytt. to left temple, syr. ferr. iodid., ol. morrh., quinine.) On third day fot. bellad. replaced by spirit lotion (on lint, re-wetted every hour). Next day all hypopyon gone. (Seton in left temple.) Ulcer healed rapidly. Cornea rather steamy.

Case 30.—(Mr. Hutchinson.) Ulceration of right cornea with surrounding infiltration and hypopyon in girl, æt. 4. Iridectomy downwards. (Fot. bellad., cinchona.) Improvement slow at first: much hastened by seton in temple.

Case 31.—(Mr. Bowman.) Central ulcer with hypopyon and slight ciliary injection in left eye of boy, æt. 2. (Fot. bellad., quinine.) Rapid improvement.

Case 32.—(Mr. Streatfeild.) Ulceration of both corneæ in delicate boy, æt. 3½. Right eye, small ulcer. Left eye, large

central onyx with hypopyon. Iridectomy upwards and onyx slit superficially. (Fot. bellad., atropine, vin. ferri; ol. morrh.) Right eye: rapid improvement. Left eye: increase of onyx: complete corneal section, followed 3 days later by paracentesis below, with partial escape of hypopyon. (Cold douche occasionally.) Slow improvement.

Case 33.—(Mr. Couper.) Small central onyx with slight hypopyon, of 5 weeks' duration in left eye of stout flabby boy, æt. 2. Lids somewhat swollen. Corneal section: no return of hypopyon. (Fot. bellad., atropine, quinine.) Rapid improvement.

Case 34.—(Mr. Lawson.) Central corneal ulcer with thick hypopyon in right eye of healthy-looking man, æt. 38. Exposed to dust and chaff. (Fot. bellad., atropine, quinine.) Steady improvement: hypopyon gone on 14th day.

Case 35.—(Mr. Hutchinson.) Corneal ulcers with infiltration and hypopyon (one-half) of 14 days' duration in left eye of girl, æt. 2, with pertussis. Sclerotic injection. Was inflamed 12 months ago. Phlyctenule in right eye. (Fot. bellad., cinchona; ol. morrh.) Hypopyon all gone on 3rd day: no return. Ulcers healed rapidly.

Case 36.—(Mr. Couper.) Superficial ulceration of entire right cornea, with thick hypopyon (one-half) of 3 weeks' duration in man, æt. 52. Has had insufficient food and clothing. Pain slight. Tension increased. (Fot. bellad., quinine, extra diet and beer.) No improvement. On 10th day paracentesis below: pus evacuated, and small piece of iris excised. Four days afterwards new epithelium began to form. No return of hypopyon except small fragment of puro-lymph at seat of iridectomy. No pain. Bare p l.

Case 37.—(Mr. Hutchinson.) Small central onyx with infiltrated margin in right eye of stout intemperate (?) man, æt. 42. Pain. Recent iritis. Iridectomy downwards. (Fot. bellad., atropine, quinine, extra diet with stimulants.) On 4th day fot. bellad. replaced by spirit lotion, followed by relief of pain. Return of acute symptoms after exposure to cold: corneal section through onyx without visible escape of purulent matter. Occasional return of severe pain. (Seton in right temple.) Slow improvement. Sent to sea-side.

Case 38.—(Mr. Streatfeild.) Right eye: sloughing ulcer of cornea in woman, æt. 29. Slight chemosis and ciliary injection.

Is suckling an infant 6 months old. (Fot. bellad., atropine, iron and quinine, beer.) Perforated on 8th day: hypopyon. Aqueous restored, and ulcer healed slowly.

Case 39.—(Mr. Wordsworth.) Left eye: extensive sloughing ulcer at lower part of cornea in lad, æt. 19. Duration 18 days: history of foreign body in eye. Aqueous muddy. Slight pain. (Fot. bellad., atropine, quinine.) Seven days later entire cornea involved, followed by irritability in right eye. Ultimately condition improved: large dense leucoma.

Case 40.—(Mr. Soelberg Wells.) Left eye: recent perforation of cornea below (from ulceration) with prolapse of iris in woman, æt. 36. Small indolent ulcer in right cornea above. Extensive lupus of face. (Fot. bellad., atropine, ferri et quin. citr.) Prolapse punctured and small iridectomy, followed by improvement. Progress tedious, and not quite satisfactory.

Case 41.—(Mr. Wordsworth.) Left eye: nearly central prolapse of iris from recent perforating ulcer in flabby boy, æt. 8. Small marginal phlyctenule. (Fot. bellad., compress bandage, quinine, syr. ferr. iodid.; ol. morrh.) Fifteen days later lower part of right cornea infiltrated with pus: formation of slough, and perforation on 4th day. (Treatment as for left.) Prolapse in left slowly flattened, and anterior chamber re-formed in both.

Case 42.—(Mr. Hutchinson.) Right eye: large, deep, perforating ulcer of cornea (below) with much infiltration, in delicate boy, æt. 5. Conjunctiva congested: lids swollen and eczematous. Impetigo of forehead and right cheek: glands in neck enlarged. Pain slight. (Ung. hydr. ox. flav. dil., seton in right temple, quinine.) On 4th day no improvement. (Spirit lotion (1 to 2), syr. ferr. iodid., and ol. morrh. added.) Two days later ulcer perforated: large prolapse of iris. Left eye now irritable, with marginal phlyctenules. (Ung. hydr. ox. flav. for left eye: discontinued in right eye.) Soon afterwards right eye became painful, and was excised: slight cellulitis followed. Left eye recovered rapidly.

Case 43.—(Mr. Streatfeild.) Right eye: interstitial suppuration of lower two-thirds of cornea of 4 days' duration. Left eye: large deep central sloughing ulcer; cornea nearly perforated; of 7 days' duration. Both painful. Woman, æt. 31. (Fot. bellad., atropine, quinine.) Central slough formed in right on 2nd day. Separation of sloughs in both eyes after a week.

Temporary relief of pain from paracentesis: repeated. Healing slow, but satisfactory.

Case 44.—(Mr. Streatfeild.) Right eye: nearly central corneal ulcer with surrounding infiltration in feeble man, æt. 70. No pain. Retention of urine (from enlarged prostate). (Fot. bellad., atropine, quinine, extra diet.) Under treatment 5 days. Slight improvement.

Case 45.—(Mr. Couper.) Left eye: ulceration of old leucoma and of surrounding cornea, with infiltration, in woman, æt. 29. Much ciliary injection and pain. Is suckling infant 8 mos. old. (Fot. bellad., atropine, quinine, extra diet.) Slight improvement. On 7th day corneal section, followed by relief from pain. Cornea cleared rapidly.

Case 46.—(Mr. Streatfeild.) Left eye: vascular ulcer near lower margin of cornea in boy, æt. 14. Cornea dull, and pitted all over; $2\frac{1}{2}$ months' duration. No pain now. (Fot. bellad., gutt. atrop. et zinci sulphat., syr. ferr. iodid., ol. morrh.) Slow improvement.

Case 47.—(Mr. Streatfeild.) Left eye: small corneal ulcer below, with surrounding infiltration, in woman, æt. 56. Right eye ulcerated 12 months ago. Considerable pain. (Fot. bellad., atropine, iron and quinine.) Rapid improvement.

Case 48.—(Mr. Streatfeild.) Left eye: central corneal ulcer, with slight infiltration, in girl, æt. 12. Crescent of minute straight vessels at lower corneal margin, swelling of lids, ciliary injection, and pain. (Fot. bellad., atropine, quinine.) Improvement quick and steady.

Case 49.—(Mr. Wordsworth.) Right eye: large, dense leucoma with marginal vascularity. Left eye: small ulcer with much corneal infiltration. Girl, æt. 7. Palpebral papillæ elevated. (Fot. bellad., ung. hydr. ox. flav. dil., syr. ferr. iodid., ol. morrh.) Slow but steady improvement.

Case 50.—(Mr. Wordsworth.) Ulceration of both corneæ in child, æt. 18 months. Fot. bellad.) Rapid improvement.

Case 51.—(Mr. Streatfeild.) Left eye: corneal ulcer with surrounding infiltration in man, æt. 47. (Fot. bellad.) Rapid improvement.

Case 52.—(Mr. Hutchinson.) Left eye: ulceration, with much corneal infiltration, of 3 weeks' duration, in girl, æt. 11. History of alternating ulceration of both corneæ. (Seton in left

temple, empl. lytt. behind ear, spirit lotion, quinine.) Rapid improvement.

Case 53.—(Mr. Hutchinson.) Left eye: small ulcer with patches of vascularity in cornea of man, æt. 23; 18 months' duration. Enlarged papillæ on lids. (Ung. hydr. ox. flav. dil., arg. nitrat. gr. x ad ʒi used once, quinine.) Benefit from seton in temple. Improvement slow.

Case 54.—(Mr. Wordsworth.) Left eye: deep, transparent, marginal, creeping ulcer, of 2 months' duration, at outer third of cornea in feeble woman, æt. 51; 18 mos. past climacteric period. Postr. synæ. (Fot. bellad. c. alumine; atropine, ferri et quin. citrat., extra diet.) Slow steady healing, beginning at the extremities, hastened by local application of arg. nitrat. gr. x (or more) ad ʒi every second morning.

Case 55.—(Mr. Critchett.) Phlyctenular ulceration of both corneæ in strumous boy, æt. 13. Much vascularity and photophobia. Lids thick with papillary elevations. (Fot. bellad., quinine, syr. ferr. iodid., ol. morrh., extra milk.) Very slow progress at first. Setons then inserted into both temples and solid nitrate of silver applied to outside of lids, followed by more rapid improvement.

Case 56.—(Mr. Hulke.) Right eye: small corneal phlyctenule in boy, æt. 14. Has had several previous attacks. (Ung. hydr. ox. flav. dil., vin. ferri, ol. morrh.) Rapid improvement.

Case 57.—(Mr. Wordsworth.) Phlyctenular keratitis in both eyes of girl, æt. 8. Much photophobia. (Fot. bellad., atropine, syr. ferri iodid., ol. morrh.) Improved slowly.

Case 58.—(Mr. Couper.) Phlyctenular ulceration of both corneæ, worse in left, in man, æt. 23. Frequent attacks. Began after measles 6 years ago. Rather anæmic. Has acne every spring. (Ung. hydr. ox. flav. dil. to right eye; fot. bellad. to left, cinchona.) Rapid improvement.

Case 59.—(Mr. Wordsworth.) Right eye: marginal phlyctenular keratitis. Left eye: phlyctenular ulcer of cornea in girl, æt. 10. Much photophobia in both. (Fot. bellad., atropine to both; ung. hydr. ox. flav. dil. to right eye; quinine, syr. ferr. iodid., ol. morrh.) Rapid improvement.

Case 60.—(Mr. Bowman.) Small vascular phlyctenular ulcers on both corneæ of delicate girl, æt. 13. Much photophobia. Similar attacks repeatedly from infancy. Pityriasis of scalp.

(Fot. bellad., atropine, calomel insufflation, syr. ferr. iodid., ol. morrh.) Steady improvement, but recovery rather tedious.

Case 61.—(Mr. Hulke.) Phylctenular ulceration of both corneæ in stout florid lad, æt. 19. Much pain and photophobia. (Fot. bellad., atropine, syr. ferr. iodid., ol. morrh., empl. lytt. frequently to temples.) Slow improvement with occasional relapses. Sent to the country.

Case 62.—(Mr. Wordsworth.) Phlyctenular ulceration of left cornea, with much vascularity and photophobia in dark complexioned, strumous girl, æt. 11. (Fot. bellad., atropine, vin. ferri, ol. morrh., quinine.) Tedious recovery.

A phlyctenule appeared on right eye, but yielded in a day or two to ung. hydr. ox. flav. dil.

Case 63.—(Mr. Wordsworth.) Left eye; marginal phlyctenules in boy, æt. 10. No pain. Slight photophobia. (Ung. hydr. ox. flav. dil.) Improved rapidly.

Case 64.—(Mr. Wordsworth.) Right eye: phlyctenular keratitis of 14 days' duration in florid strumous lad, æt. 20. Much vascularity. No pain. Deep anterior chamber. Left eye: similar condition of 3 days' duration. Slight pain. Much photophobia in both. Is subject to such attacks. (Fot. bellad., atropine, quinine.) Improved steadily.

Re-admitted 10 days after discharge with return of above condition: improved under similar treatment; sent to seaside.

Case 65.—(Mr. Lawson.) Left eye: phlyctenular ulceration of cornea in girl, æt. 19. (Fot. bellad., syr. ferr. iodid.) Steady improvement.

Case 66.—(Mr. Bowman.) Right eye: phlyctenular ulceration of cornea with infiltration and vascularity. Left eye: phlyctenular ophthalmia. Man, æt. 37. No pain. Has had two previous attacks in right (6 years ago and 9 months ago). Impetiginous eruption on chin and neck. (Fot. bellad., quinine, iron.) Steady improvement, hastened latterly by calomel insufflation.

Case 67.—(Mr. Critchett.) Left eye: phlyctenular ulceration of cornea in woman, æt. 24. Eye has been frequently inflamed since age of 2 years. Right eye affected first 2 years ago. Myopia $\frac{1}{6}$. (Fot. bellad., atropine, quinine, iron, seton behind left ear.) Rapid improvement.

Case 68.—(Mr. Wordsworth.) Phlyctenular keratitis in both

eyes of boy, æt. 9. Great photophobia. (Fot. bellad., atropine, ung. hydr. ox. flav. dil., syr. ferr. iodid., ol. morrh.) Improved steadily.

Case 69.—(Mr. Couper.) Phlyctenular keratitis of both eyes with blepharitis marginalis in girl, æt. 19. Much photophobia. Eyes have been always weak. (Fot. bellad., ung. hydr. ox. flav. dil., liq. arsenic.) Steady improvement, seemingly hastened at last by empl. lytt. to temple.

PART II.

A Periscope

OF

CONTEMPORARY OPHTHALMIC LITERATURE.

BUPHTHALMOS.

PROF. HIRSCHBERG (Graefe's Archiv., Bd. xxii, Abth. iii, p. 139) remarks that under the term congenital buphthalmos two different conditions are included. The one form is that of cornea globosa, in which, with increasing deepening of the anterior chamber, luxation of the lens, and excavation of the optic nerve, blindness ensues quite gradually, but even in childhood. In other cases of congenital and increasing enlargement of the eyeball, the condition is due to fœtal iritis (irido-choroiditis); from the first we find, without any evidence of perforation of the cornea, circular synechia and bulging of the iris associated with blindness of the affected eye. Of the latter form he narrates an early stage. The patient was a child six months old, whose mother had one normal eye, and the other highly myopic, with sclero-choroiditis and slight amblyopia. Fourteen days after birth the child's left eye was noticed to diverge, and subsequently to enlarge gradually. When seen, the left eye was markedly larger than the other, the sclerotic was white, the cornea clear, the iris bossy, the pupil small and closed with a white membrane, the tension was markedly increased, the eyelids irritated by the enlarged eyeball. After removal, the eyeball was hardened, divided horizontally, and then sections were made with the microtome. Evidences of severe iritis such as occurs after birth were found. The anterior chamber was diminished in all directions, its section had the form of a battle-axe, the broad part directed forwards to the cornea. The pupil was closed with a firm coherent membrane, intimately united with the anterior capsule of the lens. The iris was pushed forward leaving a space filled with fluid between it and the lens, apparently a real serous cyst, caused by splitting of the uveal layer of the iris into two laminæ; a representation is given. The retina and choroid seemed but slightly altered, the disc showed a deep cup, with a narrow entrance. Stress is laid on the peculiarity

of the exudation being between the two laminæ of the uveal layer of the iris.

CONGENITAL MICROPHTHALMOS.

Prof. Hirschberg (l. c.) describes a specimen which he thinks is an instance of microphthalmos, due to disease of the eyeball during fœtal life. The patient was a girl aged 14, whose right eye was said to have been blind and abnormal from birth, without any inflammation or suppuration having been noticed at any time. There was nystagmus of both eyes, and convergence of the right. The left eye was hypermetropic, but otherwise normal. The horizontal diameter of the right cornea was 7 mm. (·28 in.), that of the left $11\frac{1}{2}$ mm. (·46 in.). The cornea was transparent, except a band in the horizontal meridian. The pupil was comparatively wide, and did not react to light. A yellowish reflex came from behind it. By focal illumination a yellowish convex bladder could be seen behind the iris, ending in a pedicle posteriorly. Pressure in the ciliary region gave rise to pain. The eyeball was removed. The posterior half seemed much larger than would have been expected from the size of the cornea. The length of the eyeball was $17\frac{1}{2}$ mm. (·7 in.), the breadth of the cornea 7 mm. (·28 in.), the heighth $6\frac{1}{2}$ mm. (·26 in.). The state of parts within the eye resembled a funnel-shaped detachment of the retina. Representations are given of the two halves of the section of the eyeball. There were evidences of inflammation of the choroid, but there was no coloboma. In the anterior part of the space behind the iris was a funnel-shaped mass containing the lens, vitreous, and retina. The anterior extremity of the funnel was 2 or 3 mm. (·08—·12 in.) behind the plane of the iris; the posterior extremity passed to the disc. The lens was very small—3 mm. (·12 in.) in thickness, and 4 mm. (·16 in.) in breadth—and was displaced upwards and outwards. The capsule was nearly empty. There was a process passing from the posterior part of the mass to the fundus, and probably represented the remains of a vessel.

OPERATIONS ON THE VITREOUS.

Prof. Hirschberg (l.c.) narrates two cases, in which he removed foreign bodies from the vitreous. He had to enucleate subsequently, and gives details of the conditions found (with illustrations).

ANOPHTHALMOS, WITH CONGENITAL CYSTS OF THE LOWER EYELIDS SIMULATING ECTOPIA OF THE EYES.

A child came under Dr. Wecker's care with absence of the eyes and cystic tumours of the lower eyelids. A quantity of fœtid sebaceous matter was evacuated from the orbits. It was doubtful whether the tumours communicated with the cranial

cavity, or represented the eyeballs. They were tapped, and some fluid evacuated resembling that of the aqueous chamber rather than cerebro-spinal fluid. The results of chemical analyses are given. (Annales d'Oculistique, Mars—Avril, 1877, p. 151, and Relevé Statisque.)

CONGENITAL ABSENCE OF IRIS WITH EXCAVATION OF THE OPTIC NERVE.

Dr. Samuelson, of Manchester, sends a note of case recorded by him in Brit. Med. Journal., Nov. 7, 1863. (Zehend. Klin. Monatsbl., xv, Mai, p. 189.)

A CASE OF MALFORMATION, IRIDEREMIA, &C.

Dr. S. Klein gives a detailed description of a case in which there was irideremia, displaced lenses, absence of the zonule of Zinn, excavation of optic disc, &c. He also quotes recorded cases of irideremia, &c. The paper is too long for sufficient abstract. (Zehend. Klin. Monatsbl., xv, Jan., pp. 21, 47.)

IRIDEREMIA IN ONE EYE IN HYDROPHTHALMUS CONGENITUS.

Dr. Brunhuber gives a detailed account of a case in Zehend. Klin. Monatsbl., xv, Marz., pp. 104—116.

DERMOID OF THE CARUNCLE.

Schiess-Gemuseus records and figures a case (Zehend. Klin. Monatsbl., xv, April, p. 135).

MICROPHTHALMOS, WITH CONGENITAL SEROUS CYSTS BENEATH THE LOWER LIDS.

Dr. Talko records the case. (Zehend Klin. Monatsbl., xv, April, p. 137.)

ANOPHTHALMOS, WITH CONGENITAL CYSTIC FORMATION IN THE LOWER LIDS (WITH AN ILLUSTRATION).

Dr. Wecker. (Zehend. Klin. Monatsbl., xiv, p. 329.)

ANOPHTHALMOS CONGENITUS.

Dr. Landesberg records four cases in Zehend. Klin. Monatsbl., xv, April, p. 141.

HERPES IRIS CONJUNCTIVÆ.

Dr. Ernest Fuchs records a case under the care of Prof. Arlt. There was a membranous pellicle formed on the conjunctiva of the lids of both eyes. There was very little swelling. The mucous

membrane of the mouth and palate was also covered with patches of membrane. In the palm of the left hand were groups of spots resembling the eruption of herpes iris. The patient, a man aged 57, recovered perfectly. The results of a microscopic examination of the membranous productions is given. (Zehend. Klin. Monatsbl., xiv, pp. 333—351.)

FRAGMENT OF PERCUSSION CAP IN THE INTERIOR OF AN EYE FOR 24 YEARS—THEN IRIDO CYCLITIS—SYMPATHETIC IRRITATION OF THE HEALTHY EYE—ENUCLEATION—RECOVERY OF THE THREATENED EYE.

Dr. Meyhoefer records the case in Zehend. Klin. Monatsbl., xv, Feb., p. 75.

NEW METHOD OF PERFORMING PLASTIC OPERATIONS.

J. R. Wolfe, in Med. Times and Gazette, June 3, 1876. The operation consists in transplanting a large graft of skin from a distance. No subcutaneous connective tissue is allowed to remain, but carefully snipped off before being applied. Wadsworth records a case. (Report of Fifth International Ophthalmological Congress, p. 237. Also note in Zehend. Klin. Monatsbl., xv, Mai, p. 192).

TRANSPLANTATION OF THE CONJUNCTIVA OF THE RABBIT IN SYMBLEPHARON BY J. R. WOLFE.

Two cases are recorded in the Lancet, April 8th, 1876.

SEROUS SUBCONJUNCTIVAL CYST OF THE LOWER LID IN A WOMAN OF 21.

Prof. Dr. Laqueur records the case. (Zehend. Klin. Monatsbl., xv, Juni, p. 226.)

NEURO-PARALYTIC ULCERATION OF THE CORNEA.

Prof. Dr. Laqueur records a case. (Zehend. Klin. Monatsbl., Juni, xv, p. 228.)

ON CERTAIN IRREGULAR PARENCHYMATOUS AFFECTIONS OF THE CORNEA, CAUSING STREAKY AND BAND-LIKE OPACITIES.

E. Raehlmann writes a paper on this subject, accompanied by illustrations. The opacities can only be detected by oblique illumination, and using a second lens to magnify. In some cases the opacities are quite superficial, in others deeper. The streaks cross one another at various angles, forming a network, and at various levels. Where they cross there are dots of opacity to be seen. The rest of the cornea may be clear, or the whole may be hazy. There is generally conjunctivitis, and often iritis. The progress is slow, except where the affection is syphilitic. The

author thinks the opacities are due to an affection of the lymph passages between the lamellæ of the cornea. If injections are made into the cornea, the fluid injected can be seen with the naked eye to diverge from the point of injection in straight or curved lines, with distinct nodes or points here and there. The lines are nothing but interfibrillar spaces, and indicate that the substance of the cornea can be split up more readily in certain directions, and exudation and infiltration materials from the sclerotic and conjunctiva can also pass more readily in the same way. He thinks that the corneal affection he describes is secondary to inflammation of the sclerotic, or episcleral tissue, or conjunctiva. Though slow in its course, the disease generally passes away without leaving serious mischief behind. Though iritis is common, adhesions are infrequent. The subjective symptoms are slight. Changes in tension are common. The compressive bandage is ill-borne, whilst warm moist applications are serviceable. During exacerbation atropine is strongly indicated. (Zehend. Klin. Monatsbl., xv, Jan., p. 1.)

STRYCHNINE IN OCULAR THERAPEUTICS.

An abstract of a memoir by G. Haltenhoff is given in the Annales d'Oculistique, Sept.-Oct., 1876, pp. 185—197.

DIABETIC CATARACT—SUGAR IN THE LENS.

Dr. Teillais. (Annales d'Oculistique, Nov.-Dec., 1876, p. 238.)

ON THE ANTISEPTIC PROPERTIES OF SOLUTIONS OF ESERINE AND ATROPINE.

Herm. Schmidt-Rimpler has been making the experiment of inoculating the cornea of rabbits with the secretion from an inflamed lachrymal sac. He found that when any of this secretion was allowed to remain for 20 or 50 minutes in a solution of carbolic or salicylic acids or chlorine-water, &c., it was deprived of septic properties. On making similar experiments with eserine and atropine he found the intensity of the action diminished but not rendered aseptic. The two solutions seemed to act in much the same way. (Zehend. Klin. Monatsbl., xv, April, p. 127.)

HYOSCYAMINE.

E. Pflüger contributes a paper to the Archives of Ophthalm. and Otol., vol. v, Nos. 3 and 4, p. 333. He discusses the different preparations used, and gives the formula adopted in his own experiments. He sums up to the effect that pure hyoscyamine prepared by Dr. Stierlin, after Dammer's recipe, has an action similar to that of atropine; but it dilates the pupil more rapidly and for a greater length of time than atropine does. After many

and varied observations, he says he can fully endorse the statement of Prof. Dor, that in the lingering forms of interstitial parenchymatous inflammation of the cornea, hyoscyamine is productive of far better results than atropine. The solution used contained 1 part of pure hyoscyamine (Stierlin) in 150.

MENINGITIS AFTER REMOVAL OF GLOBE FOR WOUND—RECOVERY.

Dr. H. Meyhoefer records the case of a man æt. 24, whose right eye he removed 48 hours after an injury. A foreign body was found. In the afternoon of the following day he became restless, his temperature rose (38·5 C., 101·4 F.), and his pulse was hard and full. He had severe pain in his head. He was ordered ice to the head, quinine and calomel. On the third day he was worse; temp. 40·2 C. (104·3 F.). He was quite unconscious. Subsequently he had convulsions. On the sixth day he began to improve. His hearing was defective when sent out at the end of a month. Cases on record of death after enucleation are quoted. (Zehend. Klin. Monatsbl., xv, Feb. 1871.)

SYMPATHETIC OPHTHALMIA.

Dr. J. Hirschberg contributes a historical note to vindicate v. Ammori's claim to priority (1835) in noticing sympathetic affection of a normal eye after traumatic iritis, &c., of the other. (Archives of Ophthal. and Otol., vol. v, Nos. 3 and 4, p. 393.)

SYMPATHETIC OPHTHALMIA.

Dr. Savary records the case of a man æt. 33, who had had his right eye struck three years previously a violent blow, without, however, any external wound resulting. The eye for long remained quiescent and the vision was good, but finally the eye became painful and the sight failed. Excision of the injured eye was advised. He declined till the other became affected at the end of three years from the time of the blow. The injured eye was then soft and shrunken. After removal a bony growth was found in it. The patient had scarcely sight enough to see to go about with a stick. (Annales D'Oculistique, Sept.–Oct., 1876, p. 151.)

SYMPATHETIC OPHTHALMIA.

Dr. Adolph Alt contributes a paper on the Anatomical Causes and the Nature of Sympathetic Ophthalmia (Archives of Ophthalmol. and Otol., vol. v, Nos. 3 and 4, pp. 395—478). He gives detailed accounts of 32 eyes examined by himself, and quotes 78 others on record; the statistics, therefore, being based on 110 eyes. Tables are given summarising the conditions found in eyes enucleated for sympathetic irritation; for sympathetic iritis; irido-cyclitis; irido-choroiditis; affection of the retina; and,

lastly, affection of the cornea. Other tables show the numbers in each of these classes in which particular injuries had occurred or pathological conditions supervened. In addition, the results obtained from these statistics are dwelt on at length. In conclusion, he states that the cause necessitating enucleation in $83\frac{1}{2}$ per cent. of the cases was an injury, leaving $16\frac{1}{2}$ per cent. of the cases attributable to idiopathic inflammation. In 39 per cent. of the 110 eyes, we encounter injuries to the hard membranes with incarceration of a part of the uveal tract, *i.e.*, in $62\frac{3}{4}$ per cent. of all the injuries. The percentage of eyes setting up sympathetic troubles without presenting cicatrices in the hard membranes is very small (about 5 per cent.). In $44\frac{1}{2}$ per cent. of the cases of injury to the hard membranes, no part of the uveal tract was attached to the wound. These cicatrices are always combined with internal changes. Cicatrices and foreign bodies in the ciliary body or incarceration of it—which are usually looked upon as the most important factor in sympathetic affections—appear in only $17\frac{1}{4}$ per cent., while the affections of the ciliary body altogether amount to $76\frac{1}{2}$ per cent. The iris is changed in only 68 per cent. and the choroid in 73 per cent., thus showing almost the same number of changes in each of the parts of the uveal tract, with only a slight percentage in favour of the ciliary body. The percentage of changes in the retina and the optic nerve is remarkably great—79 per cent.; of the retina alone 73 per cent., and in 58 per cent. of these the retina is detached. Notwithstanding the fact that there is but one case described in which there was any affection of the retina without coincident affection of the uveal tract, it appears to him that the great percentage of affections of the retina and optic nerve should engage very earnest consideration—the more so as there are but $16\frac{2}{3}$ per cent. which present changes in the ciliary nerves. The author is inclined to attribute a greater share to the optic nerve in the transmission of the affection from one eye to the other, than is usually done. The sympathetic system must not be left out of view. In more than 99 per cent. of the cases, we have to deal with changes in the vascular membrane of the eye—consequently with disturbance of the entire circulatory system. It is worth mentioning that in 13 of the cases where eyes were enucleated for sympathetic iridochoroiditis, the other had been lost by panophthalmitis purulenta, and that of the five enucleated for neuroretinitis sympathetica three showed retinal detachment. The following practical deductions conclude the paper:—1. The entire nervous apparatus of the diseased eye participates in the transmission of the affection to the other. 2. Scars in the hard membranes, whenever combined with alterations in the other parts of the eye, particularly in the uveal tract, retina, or optic nerve, are capable of calling forth sympathetic affections of the other eye at any time. 3. Purulent panophthalmitis makes no exception. 4. Consequently its artificial production, as a prophylactic measure against

sympathetic affection, is reprehensible. 5. The time at which sympathetic affections most frequently manifest themselves varies between seven days and eight weeks after the beginning of the disease of the first eye. There does not appear to be any case noted in which structural changes occurred within a month. In the earlier cases, enucleation was performed for sympathetic "irritation." 6. As soon as the first trace of a sympathetic affection manifests itself (a diagnosis which should be made with the greatest care) therapeutic measures should be resorted to. 7. The only efficient means consists in enucleation of the eye first affected. 8. When it is fairly presumable that the affection has extended to the optic nerve, the removal of a large piece of the nerve, together with the globe, should not be omitted. 9. Should a case come under care in which the iris or capsule of the lens is incarcerated, the incarcerated iris or capsule should be freed before sympathetic irritation has made its appearance. In recent cases, an abscission of the prolapse, with or without iridectomy, will obviate in most cases the impending danger of sympathetic ophthalmia.

VASOMOTORY AND SECRETORY NEUROSES OF THE EYE.

Dr. Schliephake contributes some observations (from Professor Nagel's clinique) on various affections of the eye associated with diminution of tension, and dependent, he thinks, on some nervous disturbance of vascular supply or intraocular secretion. These cases have not received so much attention as they deserve, or as those attended by increase of tension. Bowman has referred to such in the course of glaucoma, and v. Graefe and Nagel have termed them essential phthisis bulbi. Nagel also has pointed out that such cases frequently occur associated merely with symptoms of slight ophthalmia. The anomaly of secretion in these cases is evidently founded on a transitory and often rapidly changing disturbance of the nerves in contrast to the phthisical softening of the globe caused by the disorganisation and partial destruction of the vessels. It is as yet completely doubtful whether the secretory anomaly is to be considered as a secretory or vaso-motor neurosis; as a phenomenon of irritation or of paralysis; as an affection of the sympathetic or of the trigeminus. Prof. Nagel has lately brought forward a number of cases in which a transitory diminution of tension—hypotony—formed a striking symptom. Graefe called attention to such diminution in chronic diffuse parenchymatous keratitis, and this has frequently been observed. There should be no complication of iritis or cyclitis. Phlyctenulæ are often associated with hypotony. A case of trachoma is narrated in which severe pain with diminished tension occurred, and was only relieved when the pupil became widely dilated with atropine. The symptoms recurred several times when the effect of the atropine had passed off. The chief

part of the paper contains examples of cases supporting the views of Prof. Nagel, advocated by him in a communication to the Heidelberg Congress in 1871. He stated that he had repeatedly observed cases of apparently mild ophthalmia, with a great diminution of tension without any considerable material participation of the external parts of the eye, especially without any change in the cornea or iris. No traumatic or other cause could be detected. Mostly only one eye was affected, sometimes both eyes alternately. In the cases recorded in the present paper there were marked local changes of temperature. In one case diminution of tension and attacks of pain were associated with lowered temperature while the pain lasted, and with raised temperature in the intervals; the bulb of the thermometer being placed on the closed eyelids or in the inner canthus, &c. The difference equalled 2°—3° C. (4° to 5° F.). In some of the cases there was moderate ciliary congestion, in others phlyctenulæ, some slight injury, &c. The chief features are the diminished tension, injection of the conjunctiva, lachrymation, small pupil, pain, and elevation of temperature. The symptoms were relieved by morphia, atropine, warm moist applications, local abstraction of blood, &c. In a few cases these eye symptoms were associated with others pointing to a partial paralysis of the cervical sympathetic (half-sided hyperæmia, &c.). In one case, "the part of the neck near the angle of the lower jaw, corresponding to the position of the upper cervical ganglion, showed a complete want of sensibility upon pressure on both sides." The paper concludes with a comparison between the symptoms met with in the cases brought forward, and those noted in undoubted affections of the cervical sympathetic. (Archives of Ophthalmol. and Otol., vol. v, Nos. 3 and 4, pp. 479—496.)

ON THE DEVELOPMENT OF MYOPIA; ON AN EASY METHOD OF MEASURING THE OPHTHALMOSCOPIC IMAGE, AND THE SIGNIFICANCE OF THIS MEASUREMENT IN ESTIMATING THE DIOPTRIC APPARATUS OF THE EYE.

By Dr. Leopold Weiss.—This paper is subdivided into six chapters, and extends over 124 pages in Graefe's Archiv., Bd. xxii, Abth. iii.

ON ASTIGMATISM OF THE CORNEA.

By Prof. Ludwig Matthiessen. (Graefe's Archiv., Bd. xxii, Abth. iii, pp. 125—130.)

ANOMALIES OF REFRACTION IN YOUNG PEOPLE.

Dr. A. v. Reuss gives a very detailed report on the state of the refraction of the eyes of a number of children in a school in Vienna. Not only are the relative conditions in one year stated

in a tabular and descriptive form, but the changes which he met with on examining the children again at the end of a year, and again at the end of another two years; that is, in 1872, 1873, and 1875. A great number of the children showed a much higher refractive condition (myopia) on testing with type than on ophthalmoscopic examination. We cannot reproduce the figures here. He found no difficulty in ascertaining the real state of the refraction by the ophthalmoscope without resorting to atropine, except in a very few. It is unfortunate that the question of treatment in the cases of so-called spasm of accommodation is not dealt with. He gives a table showing the different acuteness of vision in different cases of myopia, the degree of the latter being the same in each. He calls attention to the fact that in some cases of (apparent) myopia very little improvement may be caused by glasses till one in particular is tried, when the smallest letters may be read at once. He has occasionally met with cases where, on testing each eye repeatedly with glasses, he has obtained evidence of a much higher myopia than on testing both eyes together. He refers to Graefe's description of plesiopia as applying almost exactly to what is now called spasm of accommodation. (Graefe's Archiv., Bd. xxii, Abth. i, pp. 211—281.)

ASTIGMATISM.

On the analytical conditions of that form of astigmatic pencil in which the two focal lines are perpendicular, each to the axis of the pencil and also to each other, and on the correction of such pencil by a plano-cylindrical lens. By G. Hay, M.D. (Archives of Ophthalmol. and Otol., vol. v, Nos. 3 and 4, pp. 497—504.)

ASTIGMATISM.

In a communication to the Archives of Ophthal. and Otol., vol. v, Nos. 3 and 4, p. 505, Dr. W. G. Dennett makes some remarks on the effects of astigmatism in distorting the view of the fundus, causing a deceptive resemblance to neuro-retinitis, &c., and advocates the use of correcting cylindrical lenses in the direct examination. He has constructed an apparatus to be added to the ophthalmoscope for this purpose.

A NEW METHOD OF ESTIMATING THE REFRACTION WITH THE OPHTHALMOSCOPE.

By Schmidt-Rimpler. (Zehend. Klin. Monatsbl., xv, Juni., p. 235.)

MODIFIED OPTOMETER OPHTHALMOSCOPE.

Dr. Baumeister describes and figures an ophthalmoscope with two discs rotating on one another, and allowing of 64 combinations, the interval between each being ·5 dioptric. There is a

catch, so that the disc stops at each lens. (Zehend. Klin. Monatsbl., xv, April, p. 131.)

OPHTHALMOSCOPE FOR ESTIMATING REFRACTION.

Dr. Badal describes and figures one with two discs, placed one above the other, the edges meeting over the perforation in the mirror. The combinations allowed are 78. It is made by Roulot, and costs 32 fr. (Annales D'Oculistique, Nov.–Dec., 1876, p. 242.)

ON THE ESTIMATION OF THE ABSOLUTE REFRACTIVE POWER OF THE CENTRE OF THE CYSTALLINE LENS.

By Prof. Ludwig Matthiessen. (Graefe's Archiv., Bd. xxii, Abth. iii, p. 131.)

THE STATE OF REFRACTION AND ACCOMMODATION BASED ON AN EXAMINATION OF 4,296 EYES.

A notice of a work by Emil Emmert will be found in Zehend. Klin. Monatsbl., xv, April, p. 148.

MYXOMA OF THE OPTIC NERVE—REMOVAL OF THE GROWTH WITH PRESERVATION OF THE EYEBALL.

Dr. Emil Gruening reports the case (Archives of Ophthalm. and Otol., vol. v, Nos. 3 and 4, p. 508). The result was good. The ophthalmoscopic appearances after the operation differed from what is recorded of Knapp's case, inasmuch as no obvious change could be detected, except that the veins and arteries became almost indistinguishable from one another.

ON THE OPERATIVE TREATMENT OF VASCULAR TUMOURS OF THE EYELIDS AND ANTERIOR PART OF THE ORBIT.

Dr. H. Knapp (Archives of Ophthalm. and Otol., vol. v, Nos. 3 and 4, p. 514) in (1) cases of cutaneous or subcutaneous nævi of the eyelids, not involving the cartilage and orbit, extirpates without hæmorrhage by using Desmarre's palpebral clamp; (2) cases of cutaneous or subcutaneous nævi involving the free edge and tarsal cartilage of the lid. Their total removal by the knife, &c., could not but leave a defect in the lid often combined with eversion and its injurious consequences. In some such cases he has operated as in the first class, *i.e.*, by removing only the epitarsal part of the growth, leaving the edge of the lid and the cartilage intact, and has seen the prolongations of the nævi in the cartilage shrink and completely disappear. He would always try partial extirpation first, restraining hæmorrhage by use of clamp. He recommends, without having tried, Dieffenbach's plan of removing successive wedge-shaped portions, cicatrisation of the first wound having been allowed to take place before a second portion is dealt with; (3) cutaneous or subcutaneous vascular

tumours of the lids extending into the orbit, but leaving the tarsus and edges of the lids free. In these cases a horn spatula introduced into the conjunctival sac and pressed against the wall of the orbit checks hæmorrhage and presses the tumour forwards; (4) orbital vascular tumours involving important parts of the eyelids, tarsus, free edge, commissure. If the orbital portion be removed the palpebral will mostly shrink up.

Examples of all these forms illustrating the points of treatment are narrated.

EXOPHTHALMOS—PULSATING ORBITAL TUMOUR—LIGATURE ON THE COMMON CAROTID.

In the Periscope for Part I, vol. viii, p. 119, is an abstract of a case under the care of Dr. Oettingen of Dorpat. In Zehend. Klin. Monatsbl. June, 1876, p. 315, the conclusion of the case is given; the lad having died at the end of two and a half years. Contrary to expectation a solid tumour was found at the base of the skull in the left anterior fossa. The bone around was much eaten away. The tumour was fairly spherical, and bulged into the orbit. It was of tolerably firm consistence. It had a prolongation into the carotid canal. Microscopically the growth proved to be a fibroma. The tumour by pressure had caused hydrocephalus forming the swelling felt and seen during life. The absence of double optic neuritis is noteworthy. Photographs of the skull showing the extensive erosion are given.

BASEDOW'S DISEASE COMPLICATED WITH SARCOMATOUS TUMOUR OF THE BASE OF THE SKULL AND OPTIC NEURITIS.

Dr. Thos. Walzberg records the case of a man aged 20, who came under care for defect of sight, memory, &c., and pain in the neck and back of the head. There was well-marked but not extreme exophthalmos on both sides. The thyroid was much enlarged. The ophthalmoscopic signs were chiefly rather large veins, and swollen optic discs, which were of an opaque white colour. He died about a month after admission, and a detailed account of the post-mortem examination is given. There was a large tumour at the base of the skull, chiefly on the right side. In front the cavernous sinus was involved and the tumour projected backwards behind the sella turcica into the posterior fossa. When the base of the skull was looked at from below, the tumour presented at three points; into the pharynx, near the foramen ovale and the foramen lacerus anterius. The microscope showed the tumour to be a small-celled spindle-cell sarcoma. It was enveloped in a connective tissue capsule. The eyes were examined microscopically, and evidences of swelling and inflammatory products found. (Zehend. Klin. Monatsbl., xiv, p. 401.)

DRAINAGE OF THE EYE BY WIRE SETON.

In Wecker's "Relevé Statisque," and in Annales D'Oculistique, Mars—Avril, 1877, p. 140, will be found an account of a new plan of drainage of the eye introduced by Wecker. The statistics include 58 cases in which he has employed wire-drainage. Detachment of the retina, 27; of the choroid, 1; glaucoma (absolute), 12; (inflammatory) 2; glaucomatous keratitis, 1; glaucomatous iritis, 1; hydrophthalmos, 5; staphyloma (ciliary), 1; (corneal), 5; sclero-choroiditis (anterior), 2; keratoglobus, 1; opacity of the cornea, 1. After some preliminary remarks on the history of previous attempts at drainage and the reasons for their failure, he says there are two points still undecided, (1) the length of sclerotic included between puncture and counter-puncture (8 to 10 mm. probably, ·32″ to ·40″), and the method of fastening the wire in reference to tightness, pressure on the eyeball, &c. The wire should be drawn on at the moment of twisting it, to ensure that it should not project into the eyeball, but not tightly enough to damage the sclerotic. These remarks are made especially in connection with its application to detachment of the retina. The comprehension of the description of the procedure is facilitated by four figures. The instruments, &c., employed are (1) a curved needle, hollow, and 3 centim. in length (1·2″), analogous to the curved canula of a Pravaz syringe; (2) a thread of gold, 6 centim. (2·4″) in length, bent at its centre so as to form a loop. The ends of this thread are to be passed along the needle nearly to its point; (3) a needle holder without a catch, having a groove which will hold the needle firmly (special contrivance); (4) a small pair of forceps with branches crossing each other (serrefils); (5) forceps with large branches for twisting the wire. The plan of procedure is as follows:—1st. To apply the wire. The lids are separated by speculum, the eye directed upwards, the conjunctiva, &c., seized with fixation forceps below and outside the cornea, and the eye drawn well up. The needle is then introduced between the inferior and external recti as near as possible to the equator, and brought out again so as to include about 1 centim. (·4″) of the sclerotic between puncture and counter-puncture. As soon as the point of the needle presents through the conjunctiva (which is liable to be pushed before it rather than perforated by it) the fixation forceps are laid aside. The point of the needle is seized (without any hurry) by the fingers or the needle-holder, and whilst the extremity of the wire is then held the needle is withdrawn. The wire is thus left in position. 2nd step.—The extremities of the thread are seized carefully and crossed over the bridge of sclerotic. The small catch forceps are then used to hold the wire, the branches being so placed that one is above and the other below (not in front and behind); the forceps in fact lying in the direction of the needle

making the puncture and counter-puncture. The forceps are then allowed to hold by their own spring. 3rd. The ends of the wire are seized with the torsion forceps and twisted together. The wire is then cut off about 3 or 4 mm. (·9″ to 1·2″) from the catch forceps, and the remaining piece bent in the form of a hook. The forceps being removed, the hook is bent down on the sclerotic so as to lie along the line of puncture and counter-puncture and not project from the globe. The wire is then gently drawn by forceps near the points of puncture and counter-puncture so as to ensure more accurate adaptation. In Zehend. Klin. Monatsbl., xv, p. 91, is a further note. He alludes to seton in corneal margin. It must be shorter than the other, and a shorter and straighter needle is used. Also Note in Graefe's Archiv., Bd. xxii, Abth. iv.

ACUTE GLAUCOMA FOLLOWING A SINGLE INSTILLATION OF ATROPINE.

The case is recorded by Dr. Hugo Magnus, and the patient was a man aged 72. The atropine was dropped in to facilitate a proper examination of cataract. The right eye only was affected though the drops had been put in both. The patient had been seen on two occasions before the atropine was used, and there was no occasion to suspect any glaucomatous condition. The symptoms came on within a few hours of the drops being used. (Zehend. Klin. Monatsbl., 1876, xiv, p. 386.)

TATTOOING THE CORNEA, WITH REMARKS ON GLAUCOMA.

By Dr. J. Hock (Archives of Ophthal. and Otol., vol. v, Nos. 3 and 4, pp. 382—392). In a case of extensive leucoma after several sittings for tattooing, a thin portion of the scar was tattooed. The next day pain extended from the eye to the side of the head, there was greatly increased tension of the globe, and vision was diminished. A corneal fistula which had previously existed was reopened, and the tension at once diminished. On several subsequent occasions tension recurred, and the fistula had to be reopened. He makes some remarks on the materials used for tattooing, the method of operating, &c., and then passes on to discuss what he regards as the most remarkable feature of the case, the glaucomatous condition of the eye coming first after the twelfth sitting, and the perfect restoration of the eye by artificial reopening of the corneal fistula. "There was increased tension four times in the course of the attendance—once, indeed, in the midst of a sitting. I could each time recognise that the lenticular apparatus was thrust forward; there was distinct bulging of the middle part of the scars; the lens could be pushed to and fro between the fingers pressed upon the inner part of the scar, and was evidently closely applied to its posterior surface." In discussing the cause of diminution of the anterior chamber he dis-

putes the theory of transudation through the cornea, as that would rather relieve the tension altogether. He thinks the fluid more probably transudes backwards. He narrates a case (compare abstract of Schnabel's paper) where a succession of circumscribed corneal opacities preceded glaucoma, and he was inclined to attribute the affection to irritation of the sensitive ciliary nerves. In the case in which glaucomatous symptoms came on during tattooing, he thought the onset could only be explained by a sudden excess of secretion in the vitreous. Whether this was the primary condition or only secondary to the admitted intra-ocular venous congestion, or the reverse order of events, he thinks cannot be definitely settled; but he inclines to the first view, because there is no clear pathological relation between venous congestion and irritation of the sensitive ciliary nerves, and there are many analogies which render probable the relation between irritation of the sensitive nerves and increased secretion. He calls attention to the fact that the thin tissue closing the corneal fistula, which only a few days before the attack had repeatedly given way to the vis a tergo of the retained fluid, remained unbroken during the glaucomatous attack. This observation he thinks peculiarly significant in view of the idea we form of the effect of intra-ocular pressure in cases of ulcerative perforation of the cornea, and it also throws a strong light upon the origin of such perforation under external pressure. Perforation of the cornea in acute glaucoma is generally followed at once by advancement of the iris and lenticular system; but in this case the escape of a drop of fluid was followed by recession of the lens and the scar, an argument that it was not so much aqueous as a retro-iridal fluid that made the trouble, and that in case of increased intra-ocular tension the vitreous volume is but the least increased. The curative effect of artificial interference proves that there were none of the conditions predisposing to glaucoma, but that the effects observed were due entirely to the multitude of little wounds.

GLAUCOMA AND IRIDECTOMY.

Dr. J. Schnabel writes at considerable length on this subject (Archives of Ophthal. and Otol., vol. v, Nos. 3 and 4, pp. 342—381). He advocates the nervous character of glaucoma, and maintains that iridectomy relieves as a neurotomy does. He discusses the different views held as to the nature of glaucoma, and then passes to the various symptoms. He lays great stress on opacity of the cornea in contradistinction to opacity of the vitreous, whereas most authors consider the corneal affection insignificant. He asserts that opacity of the vitreous rarely occurs. He quotes Schweigger to the effect that the diffuse opacity of the cornea is the most apparent and important of the opacities of the different media in cases of inflammatory glaucoma, and that it is found early and constantly, while a simultaneous opacity

of the vitreous could never be demonstrated. Schnabel says he has found that when the media are too opaque to allow of the fundus being observed, there has been such a dense opacity of the cornea that the state of the vitreous could not be demonstrated, and that where the cornea was transparent he never found any opacity of the vitreous. The corneal opacity is most frequently found in the pupillary region. He narrates two cases where attacks of corneal opacity and dimness of sight occurred with increase of tension, but without any excavation, and in which the symptoms finally disappeared without an operation being performed. He also describes a similar series of attacks in a patient before iridectomy was performed for glaucoma. He also mentions a case where attacks of dimness occurred after iridectomy, but without any visible changes. He quotes a case recorded by Schiess in which dense opacity of the cornea had existed about five months before operation, and yet the cornea became quite clear subsequently. Schnabel regards the corneal opacity as due to secretion of a turbid fluid, as a neurosis of the secretory nerves. The pain present in glaucoma differs from that met with in inflammatory affections. A trigeminus neuralgia has preceded glaucoma (Hutchinson, Wegner, Hippel). Schnabel had a case where neuralgia of the trigeminus was the first symptom of glaucoma. The neuralgia is not the cause, but the consequence, or rather a symptom of glaucoma. The episcleral congestion will not alone avail as proof of inflammation. In reference to increase of tension and its cause, he discusses the various affections in which it occurs, and arrives at the conclusion that there must be distension or stretching of the ring at the sclero-corneal junction. He passes on to consider the opposite class of cases, where diminished tension occurs, in detached retina for instance, and attributes these to nervous disturbance. He sums up that the regulation of the conditions of secretion and absorption—the compensation in cases of accidental disturbances—belongs to the domain of nervous action, and a disturbance of this action must be looked upon as the cause of the permanent increase of the vitreous humour in cases of glaucoma, and of the permanent diminution of this fluid in cases of detachment of the retina and essential phthisis. The iridectomy reaches only those symptoms of glaucoma which are the expression of the neuropathy, and the more pronounced these are the more brilliant will be the effect of the operation. As to what nerves are diseased, we can only say that necessarily the fibres of the trigeminus are affected, and that an irritation of the fibres of the sympathetic is rendered very probable by the dilatation of the pupil; but we are not in a position to declare whether the increased tension depends upon the irritation of the trigeminus or of the sympathetic. It seems immaterial what part of the iris is removed, but it is certain that iridectomy remains ineffective when the incision lies in the cornea. It must be commenced beyond the sclerotic margin. It is very probable that we

have to look for the seat of the starting point of the morbid condition in the peripheral portion of the chamber.

Wecker and Stellwag consider that the scleral cicatrix possesses properties which render the further progress of the disease innocuous. Such explanations do not cover the whole of the ground, they attempt only to make intelligible the diminution of the tension. The iridectomy also abolishes other morbid phenomena independent of the tension, so that such theories lack in comprehensiveness. Many points of interest are touched on in the course of the elaborate examination of the symptoms connected with glaucomatous affections which cannot be noted in an abstract.

GLAUCOMA.

Dr. Max Knies contributes a paper on glaucoma to Graefe's Archiv. f. Ophth., Bd. xxii, Abth. iii, p. 163. He gives an account of fifteen specimens of glaucomatous eyes in Prof. Becker's collection, with illustrations. 1. Glaucoma absolutum (total blindness from chronic glaucoma). 2 and 3. The same. 4 and 5. Glaucoma inflammatorium. 6. Retinal detachment, with consecutive choroiditis glaucomatosa, and hæmorrhage. 7. Amaurosis after glaucoma apoplecticum. 8. Cataracta tremulans, with detachment of the retina and secondary acute glaucoma. 9. Chronic glaucoma with corneal affection, hypopyon, &c. 10. Cataract, preliminary iridectomy followed by iritis and choroiditis glaucomatosa. 11. Amaurosis after glaucoma absolutum. 12. Glaucoma apoplecticum with staphyloma corporis ciliaris. 13. Glaucoma apoplecticum. 14 and 15. Chronic glaucoma. In some general remarks on the series he states that the pathological condition he found most constant, and in his opinion the most important one, was adhesion between the peripheral portion of the iris and the cornea, causing obliteration of the peripheral portion of the anterior chamber (Fontana's space). This was often due to a cellular growth between the iris and cornea, and could not, therefore, depend merely on the iris being pressed against the cornea. Occasionally this growth had contracted so as to shorten the anterior surface of the iris, and cause the pigmented posterior surface to be drawn forwards at the pupillary margin and present in front. In association with obliteration of Fontana's space there was invariably cell growth in and around the canal of Schlemm. The ciliary body and the subconjunctival tissue, and also the central part of the iris may likewise be infiltrated. In acute cases this almost always occurs; in others, and especially in the chronic ones, the inflammation may be strictly limited to the neighbourhood of Schlemm's canal. As this locality is the chief seat of the exit of fluid from the interior of the eye, and especially from the vitreous, it may easily be the seat of inflammation independent of any other part, and obliteration of the space of Fontana must lead to increase of the intra-ocular pressure. Further, if we establish the

fact that the cardinal symptom of glaucoma, increased intra-ocular pressure, is the necessary consequence of an indurating inflammation in the region of the canal of Schlemm, we shall also find that other symptoms, hitherto regarded as consequences of the pressure, may be more easily and satisfactorily accounted for. These are opacity of the cornea, anæsthesia of the cornea, iridoplegia, apparent shallowness of the anterior chamber, paralysis of the accommodation, and congestion of the veins of the anterior part of the sclerotic. The constancy of the last symptom in contrast to the uncertainty of the former ones also is in favour of glaucoma being due to an inflammation in the region mentioned. The corneal symptoms may readily be explained by the fact that owing to the obliteration of Fontana's space, passage of the aqueous occurs through the cornea in abnormal quantity, and interferes with its functions. This applies especially to acute cases, and as the cornea is only infiltrated with serous fluid, it may recover as soon as the cause is removed. In acute cases iridoplegia is rarely absent; in chronic ones, on the contrary, it may be wholly wanting. An inflammatory process, resulting in adhesion of the periphery of the iris, as is common in the chronic cases, may leave a mobile pupil. Increased pressure does not prevent conduction along nerves otherwise unaffected. The shallowness of the anterior chamber is probably in some measure only apparent, for the lens in the cases examined was not pushed forwards at all. The shallowness of the anterior chamber, that is the advance of the pupil, cannot be regarded as a necessary consequence of increased pressure, for injection of fluid into the vitreous caused as much tension in front as behind the lens, and the latter remained in position. In one of the cases, in which inflammatory glaucoma was present in one eye, inflammatory changes were found in the other eye around the canal of Schlemm, and there was also distinct "choked disc" (Stauungs papilla). It would appear, therefore, that in inflammatory glaucoma, at least, the choked disc precedes the stage of excavation. This is plausible, because increase of tension would obstruct the outflow of blood, and induce venous congestion of the retinal vessels, and hence prominence of the disc. This condition is only of short duration, however, but leads to diminished nutrition, and the latter in the later stages may cause cataract. The author does not wish to assert that every case of glaucoma can be explained by inflammation around Schlemm's canal, and adhesion of the iris and cornea, but in all the cases he has examined these conditions were present. Glaucoma is more common in hypermetropes than in myopes, and the shallow anterior chamber of the former would facilitate the formation of adhesion between the iris and cornea. The shallow anterior chamber of old persons would also aid the same process, though here other causes may come into play. Anterior synechiæ also, for the same cause, are more dangerous than posterior ones. He proceeds to discuss other theories of the causation of glau-

coma. He regards the inflammation around the canal of Schlemm as primary, and not merely secondary to increased ocular tension. He takes as the type of glaucoma the so-called chronic form. Obliteration of the space of Fontana of course leads to increased tension. The cornea can ill supply the increased demand for transudation. Associated with increased tension we also have diminished nutrition, and these two conditions lead to changes in the disc and lens. If there is added further increase of tension owing to nerve influence, then an attack of glaucoma results. In operating for glaucoma the author agrees as to the necessity for a peripheral incision. He suggests that possibly good results might follow incising the sclero-corneal junction from the anterior chamber subcutaneously, so to speak, by means of an instrument resembling a discission needle. Prolapse of the iris would be avoided more certainly than by any other procedure, and the troublesome retinal hæmorrhages which so frequently occur after iridectomy would be less common. His own cases afford no means of judging as to the effects of iridectomy, as the operation had only been performed in two.

TUMOUR OF THE CHOROID.

Dr. J. Hirschberg details the careful microscopic examination of a case of sarcoma in which the retina was also affected. Illustrations are given. Owing to the mode of cutting adopted, he was able to obtain sections of the parts *in situ* thin enough for microscopic examination. (Graefe's Archiv., Bd. xxii., Abth. i, p. 135.)

COLOBOMA CHOROIDEÆ OF EACH EYE WITHOUT ANY AFFECTION OF THE IRIS.

Prof. F. J. von Becker records a case with illustrations. He also notes a case in which one eye only was affected. (Graefe's Archiv., Bd. xxii, Abth. iii, p. 221.)

RETINAL HÆMORRHAGES AND MELANÆMIA AS SYMPTOMS OF AGUE.

By Dr. Stephen Mackenzie (Medical Times and Gazette, June 23rd, 1877, with illustrations).—The first case noted was that of a man aged 30. When admitted into the London Hospital, suffering from ague, recent hæmorrhages were found in the retina of each eye. They presented the streaky appearance so often assumed by hæmorrhages in this situation, and were chiefly seen along the course of the larger vessels, especially arteries which they in places obscured. Sprinkled about the fundus, and most numerous near the disc, were many small round bright spots, resembling pinholes pricked in a piece of paper held up against the light. The hæmorrhages faded away gradually. As each died

away, it left to mark its former situation one of the shiny white spots of which mention has been made. There was no albumen in the urine. The blood contained pigment. Subsequently a fresh hæmorrhage of a different character was detected. It was round in shape, with a sharply defined margin, and was of a uniform dark red colour. It was not at all streaky, like the others previously observed. It gradually altered in colour, the centre becoming first purple and then yellow, and much paler than the circumferential portion, whilst the latter became as it were frayed out. Other cases of ague at the Seamen's Hospital at Greenwich were examined, and hæmorrhages were found in two cases out of six who happened at that time to be in the hospital. The hæmorrhages are probably found in the early stages and acute cases. None of the patients complained of loss of sight.

PURPURA—PERIVASCULAR RETINAL HÆMORRHAGES—RECOVERY.

A case is recorded in the Medical Times and Gazette, March 3rd, 1877, under the care of Dr. Stephen Mackenzie. The patient was a lad aged 8. On admission there were spots of purpura on the extremities. There were no changes in the fundus oculi. Subsequently, Mr. Couper reported that "on a main branch of a retinal vein immediately above the disc are two minute portions, which appear, from their colour, comparatively empty of blood. They are bounded by normally filled portions of vessel. This seems due to the fact that the vessel is encircled by a collar of blood, beyond which is the pale portion of vessel. It must also be due in part to slight grayness of the retina over that part of the vessel." There was no reason to suspect embolism. There were four such spots altogether. All the large vessels had thickened sheaths. The appearances were similar in the two eyes. A few days later similar spots were found in the arteries, and one in the disc close to the porus opticus. The retinal hæmorrhages persisted long after all had disappeared from the skin. Dr. Mackenzie in some remarks appended to the case argues that the purpura was of nervous origin.

HÆMOPHILIA—PURPURA—RETINAL HÆMORRHAGES—PURPURA OF RETINA—DEATH.

In the next number of the journal Dr. Mackenzie publishes a case under the care of Dr. Andrew Clark. The patient was a girl aged 13. She had showed a considerable disposition to bleed from slight causes. Purpuric spots and bruises were noticed in various parts. At first there were no changes within the eyes, but a few days subsequently a large hæmorrhage was found in each eye near the disc and obscuring vessels. She died about a month after admission. The autopsy was forbidden.

THE COMMUNICATION BETWEEN THE RODS AND CONES AND THE INNER NUCLEAR LAYER OF THE RETINA, AND BETWEEN THE LATTER AND THE GANGLION CELLS.

Dr. R. Marcus Gunn contributes a paper to the Journal of Anatomy and Physiology (vol. xi, Part iii, p. 357), in which he describes and figures a mode of communication he believes he has discovered between the inner nuclear layer of the retina and the rods and cones and between the ganglion cells and the inner nuclear layer. From the study of sections of the human retina prepared with osmic acid, and subsequently hardened in alcohol, he has been able to trace the outer process of one of the bipolar cells of the inner nuclear layer through the external molecular layer immediately into the bulb of a cone-fibre. (Illustrations are given.) The connecting fibre, which is often tapering, generally passes to one of the edges of the bulb. It is sometimes comparatively broad and ill-defined, at other times finer but much more distinct, and may then take a somewhat wavy course, and have a more oblique direction; but it is possible these differences of appearance depend upon the aspect from which the connecting fibre happens to be viewed. Occasionally, but rarely, the bulb of a cone-fibre has seemed to receive two such processes, one at either side, and it is not unlikely that this may be the normal state of the case in all, the second connection having been severed in making the sections. In some instances the outer process of the inner nucleus bifurctaes close to or within the substance of the external molecular layer. Mr. Hulke has shown that two separate fibres pass outward from the inner nucleus instead of a single bifurcating process. It has occasionally been possible to follow one at least of the resulting branches into the bulb of a cone fibre. Whether the other branch ends in a cone fibre or a rod fibre is doubtful. It seemed to split up into fine branches. In a solitary instance a direct communication was thought to have been seen between a branch of one of the ganglion cells and the inner end of one of the internal nuclei, and this nucleus again was by its outer process connected with the bulb of a cone-fibre. The cacao butter method of imbedding was employed. Great difficulty was experienced in making the plane of section exactly vertical to the surfaces and corresponding with the direction of the inclination of the fibres of the retina.

Prof. Fr. Merkel has been working at the same subject, and his observations seem to confirm the above. His paper is in Bd. xxii, Abth. iv, of Graefe's Archiv., p. 1–23. He has arrived at his results not by making sections but by tearing. He uses osmic acid and an alcohol-glycerine watery solution. He thinks the only way of arriving at certainty is by tearing out a single preparation reaching from the rods and cones to the inner nuclear layer. He wanted a macerating solution which would not

destroy the connection between the outer and inner parts of the retina, but would render the outer molecular layer transparent enough to show these processes. He finds the cone-fibres have sheaths, and the lower end terminating at the outer molecular layer when exposed to view shows a section as of an axis cylinder and its white substance. These appear as rings when the outer molecular layer is looked at from the surface, not in section. In section they look like an interrupted line—a series of dashes. The fibres connected with the rods are so delicate that it was difficult to trace them. He believes he has traced fine processes however from the inner nuclear layer to the rods. He has not paid much attention to any connection between the ganglion cells and the inner nuclear layer, looking on the matter as a foregone conclusion, but he has seen many fine processes which he has no doubt go to the inner nuclei. The processes pass a long way obliquely or horizontally through the inner nuclear layer. The author concludes with remarks on the framework of the retina.

THE HUMAN RETINA.

Fr. Merkel contributes a paper giving a summary of our present knowledge in Zehend. Klin. Monatsbl., xv, Juni, p. 205—226.

GLIOSARCOMA OE THE RETINA FOLLOWING AN INJURY.

The patient was a lad aged 15. The other eye was threatened with sympathetic inflammation when the globe was removed. A section of the globe is shown. A detailed account of the history and histology of the specimen is given by Dr. Walzberg (Zehend. Klin. Monatsbl., xv, Mai, p. 172—189).

RETINAL PULSATION.

Dr. Jos. Jacobi in the first of a series of papers on the circulation within the eye deals with the retinal pulsation. In reference to the *venous* pulsation, he says: (1.) It is of no pathological importance, being purely physiological. (2.) The diminution of the vein is synchronous with the cardiac systole. (3.) The phenomenon is only perceived on the disc, or at any rate within its immediate neighbourhood. Occasionally, at the same time that the vein diminishes in size, a distinct enlargement of the part of the vein close to and on the peripheral side of the pulsating portion may be detected. (4.) Excepting in the retinal veins, no such phenomenon is observable within the eye. The choroidal veins especially show no rhythmical alteration in colour. He then discusses the theories of Coccius and of Donders in reference to the production of this pulsation. The former holds that the sudden influx of arterial blood exercises pressure most easily on

the veins, and the outflow of blood by them is therefore expedited. Donders thinks the pressure is exercised through the vitreous on the parts where the lateral blood pressure is least, that is, the large venous trunks, and that in consequence they will be compressed and the outflow of blood hindered. The choroidal vessels are said to be protected by peculiar anatomical conditions. The fact that increased pressure on the part of the vitreous never *per se* favours the exit of blood, but on the contrary retards it, is opposed to Coccius' theory. Against the second theory may be urged the necessity of supposing a peculiar anatomical condition of the choroidal veins. The limitation of the pulsation is difficult to explain on either theory. The author is inclined to look as Graefe and Jäger did, to conditions met with in the disc and nerve as the proximate causes of the phenomenon. He then describes the anatomy of the physiological cup and the course of the vessels. It is when they curve sharply, forming a point or knob, or there is a kinck that spontaneous venous pulsation is generally seen. The physiological cup is the part which is most likely to yield to pressure. If, therefore, there really is any systolic increase of pressure on the vitreous, the shape of the cup will be altered. It will be deepened and widened. This alteration will not affect the arteries perceptibly on account of their thicker walls and the blood pressure; whilst the veins will be easily influenced. They will be elongated and stretched and pressed laterally, and at certain parts bent sharply, and thus an additional impediment to the circulation will be afforded, and their calibre diminished. The central portion of the vein must collapse. The degree of the collapse will depend on the abruptness of the bend and the increase in the intra-ocular pressure. Immediately after the cessation of the dilatation of the arteries, the elasticity of the disc will enable it to resume its former shape, and the obstruction to the venous flow will be removed. He gives his reasons for believing the disc to be the "locus minimæ resistentiæ." In favour of his hypothesis he quotes the results of ophthalmoscopic examination. In addition to this so-called pulsation, which is really not a pulsation, but a phenomenon *sui generis*, a true pulsation may be met with. In a case recorded by Reynaud, the retinal veins pulsated to their smallest branches. Another and peculiar case is recorded by Becker. In reference to the *arterial* pulsation, he remarks that it is generally taught that there are two forms, a true pulsation met with in cases of insufficiency of the aortic valves, &c., and also a mere intermittent flow of blood into the eye (glaucoma, &c.), where there is increased pressure within the eye. The first is characterised by systolic enlargement or elongation (movement) of the arteries, the other by diastolic pallor of the disc. The author doubts whether these two forms are sharply separated. The eye of an albino rabbit is well adapted for observation. He thinks both forms come under the designation of a true arterial pulsation, and

in each the pulsation consists essentially in a continuation of the systolic arterial wave into the ophthalmoscopic field. Pressure on the eyeball not only produces local anæmia, but the obstruction to the circulation causes increased blood pressure and pulsatile movements in the different arteries before their entrance into the eyeball. These increased pulsations are continued for a certain distance within the globe. (Graefe's Archiv., Bd. xxii, Abth. i, p. 111—134.)

GLIOMA OF THE RETINA AND OF THE OPTIC NERVE.

Dr. Baumgarten narrates in detail the history, &c., of a child with glioma, under the care of Dr. Jacobson. The microscopic examination of the enucleated eyeball is entered into fully, and the question discussed whether the growth was primarily of the retina, or of the nerve. (Graefe's Archiv., Bd. xxii, Abth. iii, pp. 203—220.)

RETINITIS PROLIFERANS.

Under this term, Prof. W. Manz records three instances of a very remarkable state of things in the fundus oculi, of which he gives two illustrations, and which has also been represented in Pl. LV of Jæger's large atlas. Here, however, it is regarded as an affection of the vitreous. In Prof. Manz's cases there were opacities in the vitreous; but the chief change consisted in whitish, filmy opacities, apparently retinal, extending in a direction chiefly upwards and downwards, in the neighbourhood of and involving the disc. The retinal vessels were partially but not wholly concealed. (A further abstract will probably be given in another number.) (Graefe's Archiv., Bd. xxii, Abth. iii, pp. 229—275.)

RESULTS OF EXAMINATION BY SPECTRUM ANALYSIS IN A CASE OF COLOUR BLINDNESS.

Dr. W. Camerer reports a detailed examination of his own perception of colour in the spectrum. (Zehend. Klin. Monatsbl., xv, Feb., pp. 51—60.)

HEMIOPIA AND AFFECTIONS OF THE OPTIC NERVES.

Prof. Schweigger (Graefe's Archiv., Bd. xxii, Abth. iii, pp. 276—323) gives details of 29 cases of lateral hemiopia, with diagrams of the more peculiar fields of vision, and of 11 other cases of various defects of the field of vision. In two cases there was a defect of the upper half of the field in each. In three cases there was limitation to a very small central field. In the first of these, the discs were white, and the arteries diminished; in the second, there was atrophy with excavation, but without increase of tension; in the third, there was atrophy of both optic nerves. In two other cases of atrophy there were defects of field limited by

line passing through point of fixation. Defects of the temporal half of each field are not uncommon in affections of the optic nerve. He gives two cases. One case of defect of the nasal half of each field is detailed, and another in which the lower half of one field was wanting in an affection (partial) of one optic nerve. Though all trace of any affection of the optic nerve may have disappeared, the defect of the field may persist. Cases of symmetrical defect of field with visible changes in the fundus should not be grouped under "hemiopia." The latter name should be restricted to defects coming on suddenly, and without any ophthalmoscopic changes, and mostly in connection with apoplexy.

ABSOLUTE AMAUROSIS FOLLOWING INJURY WITHOUT OPHTHALMOSCOPIC CHANGES.

The patient was 17 years of age. He received a blow from an iron bar on one eye. The sole defective sign was dilatation of the pupil. There was no perception of light. (Prof. Dr. Laqueur, Zehend. Klin. Monatsbl., Juni xv, p. 231.)

AMBLYOPIA WITHOUT OPHTHALMOSCOPIC CHANGES—RETRO-OCULAR NEURITIS.

Herm. Schmidt-Rimpler records the case of a young woman who had had almost complete amaurosis for more than a year. Strychnine injections were tried without effect; then applications of the artificial leech. The pupils began to act, and then hyperæmia of the disc was noticed. Inunction of mercury was tried. Ultimately complete recovery resulted. In another case, the patient a man aged 28, great defect of sight was associated with only slight œdema and hyperæmia. Subsequently well marked atrophy developed, but the sight improved. (Zehend. Klin. Monatsbl., xv, p. 165.)

AMAUROSIS IN CONSEQUENCE OF LOSS OF BLOOD.

Dr. Landesberg records one case of double amaurosis from hæmatemesis, one case of monocular blindness from metrorrhagia, and another from epistaxis. In the first case, the patient was not seen for 18 months. The discs were in a state of advanced atrophy. In the second, the affection was not noticed for a fortnight after the loss of blood. There was partial atrophy. In the third case, the eyes were examined on the following day. Both eyes were then in a state of slight neuro-retinitis, but the one recovered whilst the other developed well-marked neuro-retinitis with hæmorrhages, and subsequently passed into atrophy. (Zehend. Klin. Monatsbl. xv, Feb., p. 95.) See *Periscope*, vol. viii, Part ii, p. 386.

SYMPATHETIC INFLAMMATION SET UP BY AN ATROPHIED GLOBE AFTER GONORRHŒAL OPHTHALMIA.

A girl aged 18, had gonorrhœal ophthalmia of both eyes, ending in complete recovery of one and atrophy of the other at the end of a month. In another fortnight, she remained in same state, but in another month (ten weeks after ophthalmia), both eyes were painful, and the recovered eye showed serous iritis. In the atrophied globe, there was extensive prolapse and adhesion of the iris which had set up cyclitis. Enucleation of this globe and the thorough use of atropine ended in almost complete recovery. The case is recorded by Dr. Meyhoefer in Zehend. Klin. Monatsbl., vol. xv, März, p. 102.

ON THE ACUTENESS OF VISION IN RELATION TO THE ILLUMINATION.

Anton Posch, of Innsbruck, contributes a paper on this subject to the Archives of Ophthal. and Otol., vol. v, Nos. 3 and 4, p. 297-332.

THE ANATOMY OF THE EYES OF ARTHROPODA.

By Dr. H. Grenacher.—Appendix to the Mai number of vol. xv of Zehend. Klin. Monatsbl. (with illustrations).

AN HISTORICAL ACCOUNT OF THE VARIOUS REPRESENTATIONS GIVEN AT DIFFERENT TIMES OF THE ANATOMY OF THE HUMAN EYE, WITH ILLUSTRATIONS RANGING FROM PEYLIGK, 1499, TO SÖMMERING, 1801.

An appendix to the Juin number of vol. xv of Zehend. Klin. Monatsbl., by Dr. Hugo Magnus.

ON VISUAL SENSATIONS.

A long critical review of a series of papers by Hering is given in the Annales D'Oculistique, 1876, Juillet–Août, p. 54. In the number for Sept.-Oct. is a detailed account of papers on the Physiology of the Retina, by Dobrowolski and A. Gaine.

RETINITIS PIGMENTOSA.

An article by MM. Warlomont et Dumez on this subject from the Dict. Encyclop. des Sciences, Méd., is given in the Annales D'Oculistique, Sept.-Octob., 1876, p. 113.

LIST OF PAPERS IN THE REPORT OF THE FIFTH INTERNATIONAL OPHTHALMOLOGICAL CONGRESS.

On the use of a Naso-Buccal Flap for Blepharoplasty.—By Henry D. Noyes, M.D. (with illustrations), p. 6.

Chronic Optic Neuritis. By W. A. Gowers, M.D., p. 14.

Coloboma Lentis. By Albert G. Heyl, M.D., p. 16.

Additional Means of Relieving Pressure of the Eyelids in Diseases of the Cornea.—By Henry D. Noyes, M.D., p. 29 (with illustration).

On Sympathetic Neuro-Retinitis.—By Adolf Alt, M.D., p. 37.

On Orbital Tumours.—By W. Knapp, p. 51.

A case of Choked Disc under Observation three years and a-half without Change in Appearance or Progressive Impairment of Vision.—By Arthur Matthewson, M.D., p. 63.

The Introduction of the Meter Measure for the Determination of Lenses.—By Prof. A. Nagel, p. 66.

The Detachment of Posterior Synechiæ.—By B. Joy Jeffries, M.D., p. 66.

The Optical Error of Conical Cornea with Report of two Cases treated by Operation.—By Henry D. Noyes, M.D., p. 72.

Ophthalmoscopes with the Metric System.—By E. G. Loring, M.D., p. 81 (with illustrations).

The Halo round the Macula Lutea.—By E. G. Loring, M.D., p. 86 (with illustrations).

Hypermetropic Squint and Insufficiency of the Internal Recti.—By E. Hansen, M.D., p. 103.

Operation for Cicatricial Entropium.—By John Green, M.D., p. 126.

On Bony Formation in the place of the Lens.—By P. De Keyser, M.D., p. 131.

Trachoma as influenced by Race.—By Swan M. Burnett, M.D., p. 134.

The Relations of Blepharitis Ciliaris to Ametropia.—By D. B. St. John Roosa, M.D., p. 137.

Analysis of 1,079 *recorded cases of Ametropia and Muscular Weakness, with Deductions respecting Asthenopia.*—By Henry D. Noyes, M.D., p. 148.

Analysis of 1,060 *cases of Asthenopia* under care of Dr. Agnew, p. 214.

On Treatment of Asthenopia by means of Regular Systematic Exercise.—By E. Dyer, M.D., p. 227.

Case of Ectropion cured by Transplantation of a large piece of Skin from the Fore-arm.—By O. F. Wadsworth, M.D., p. 237.

A convenient method of applying Warmth to the Eye.—By Dr. F. Buller, p. 240.

Remarks on the Ciliary Muscle.—By E. G. Loring, M.D., p. 243 (with illustrations).

Sub-conjunctival Cysticercus.—By Charles J. Kipp, M.D., p. 250.

On Inherited effects of Lesions of the Sympathetic Nerve and Corpora Restiformia on the Eye.—By Dr. Dupuy, p. 252.

Filamentous Body present in the Anterior Chamber.—By Dr. Argyll Robertson, p. 256 (with illustration).

Case of Recurring Sarcomatous Tumour of the Orbit in a Child; extirpated for the third time, and ultimately causing the death of the patient.—By Dr. Thomas Hay, p. 258 (with illustrations).

Metrical Ophthalmoscope.—By H. Knapp, p. 263.

PAPERS IN ANNALES D'OCULISTIQUE FOR JAN.-FEV. AND MARS-AVRIL, 1877.

F. C. Donders.—*A Developmental Explanation of the Ocular Movements*, pp. 5 and 97.

Teillais.—*Three cases of Rupture of the Choroid* (with woodcuts), p. 26.

L. de Wecker.—*The use of Ésérine in Ophthalmic Practice*, p. 31.

Warlomont et Duwez.—*Etiology of Neuro-Retinitis*, p. 114.

TITLES OF PAPERS IN GRAEFE'S ARCHIV., BD. XXII, ABTH. IV.

On the Histology of the Lens.—By Ritter, p. 26.

On Binocular Indirect Vision.—By Dr. W. Schön, pp. 31—62 (with illustrations).

Results of the Examination of the Eyes of School-children at Luzerne.—By Dr. Pflüger, pp. 63—117.

On the Theory of Vision.—By J. Hirschberg, pp. 118—125.

Cases of Cysticercus Cellulosæ in the Fundus of the Eye. With illustrations, pp. 126—135.

Contributions to the Pathological Topography of the Eye.—By J. Hirschberg. No. iii. *Sympathetic Irritation and Inflammation*, pp. 136—146.

Supplement to a Paper "Studies on the Field of Fixation," in former number (see *Periscope*, vol. viii, part iii, p. 623).—By Dr. Schneller, pp. 147—156.

Clinical Contributions to the Study of Glaucoma, and especially to our knowledge of the Mode of Origin of the Pressure Excavation.—By Dr. S. Klein, pp. 157—208.

Glaucoma and Drainage of the Eye.—By Dr. Wecker, pp. 209—14.

On Calabar Bean and its Therapeutic Employment.—By Dr. Adolph Weber, pp. 215—232.

On Detachment of the Retina and its Causes.—By E. Raehlmann, pp. 233—252.